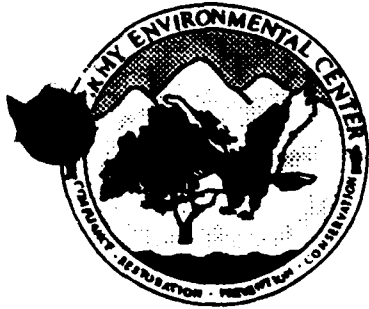


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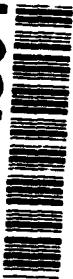
FINAL  
REMEDIAL INVESTIGATION REPORT  
APPENDIX  
DATA ITEM A009

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APPENDICES M THROUGH R  
VOLUME 7 OF 7

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**REMEDIAL INVESTIGATION  
BADGER ARMY AMMUNITION PLANT**

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**REMEDIAL INVESTIGATION  
BADGER ARMY AMMUNITION PLANT**

**APPENDICES**

(continued)

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**APPENDIX M**  
**CALCULATIONS FOR PARAMETERS USED IN RISK ASSESSMENT**



**SECTION 1**  
**COMPARISON OF PREDICTED DAILY INTAKES**  
**WITH ALLOWABLE DAILY INTAKES FOR ESSENTIAL NUTRIENTS**

**REMEDIAL INVESTIGATION**  
**BADGER ARMY AMMUNITION PLANT**

SCENARIO (MEDIUM) AREA	COMPOUND	MAXIMUM DETECTED CONCENTRATION ( $\mu\text{g}/\text{mg}$ )	PREDICTED INTAKE ( $\text{mg}/\text{kg}/\text{day}$ )	ALLOWABLE DAILY INTAKE <sup>1</sup> ( $\text{mg}/\text{kg}/\text{day}$ )	BASIS FOR ALLOWABLE DAILY INTAKE <sup>1</sup>
Construction Worker (subsurface soil)					
Propellant Burning Ground	CA	104,249	0.0006	60	RDA
	MG	59,777	0.0003	10	RDA
Final Creek Outflow	FE	13,900	0.00007	10-20	RDA
Oleum Plant and Pond	FE	43,600	0.0002	10-20	RDA
Older Child Exploring (Sediment)		( $\mu\text{g}/\text{mg}$ )			
Oleum Plant and Pond	CA	36,900	0.002	60	RDA
	NA	120	0.000007	31	Estimated adequate and safe intake
Older Child Exploring (Surface Water)		( $\text{mg}/\text{l}$ )			
Rocket Paste Pond	CA	38,200	0.9	60	RDA
	FE	31,700	0.8	10-20	RDA
	K	44,000	1.1	154	Estimated adequate and safe intake
	MG	20,900	0.5	10	RDA
	NA	2,000	0.05	31	Estimated adequate and safe intake
Nitroglycerine Pond	CA	15,200	0.4	60	RDA
	FE	3,970	0.1	10-20	RDA
Nitroglycerine Pond (continued)	K	15,000	0.4	154	Estimated adequate and safe intake

SECTION 1  
COMPARISON OF PREDICTED DAILY INTAKES  
WITH ALLOWABLE DAILY INTAKES FOR ESSENTIAL NUTRIENTS

REMEDIAL INVESTIGATION  
BADGER ARMY AMMUNITION PLANT

SCENARIO (MEDIUM) AREA	COMPOUND	MAXIMUM DETECTED CONCENTRATION	MAXIMUM PREDICTED INTAKE (mg/kg/day)	ALLOWABLE DAILY INTAKE <sup>1</sup> (mg/kg/day)	BASIS FOR ALLOWABLE DAILY INTAKE <sup>1</sup>
Ballistics Pond	MG	5,880	0.1	10	RDA
	NA	8,320	0.2	31	Estimated adequate and safe intake
	CA	6,510	0.2	60	RDA
	FE	315	0.01	10-20	RDA
	K	1,940	0.05	154	Estimated adequate and safe intake
	MG	2,920	0.07	10	RDA
	NA	3,780	0.09	31	Estimated adequate and safe intake

## Notes:

RDA - Recommended Daily Allowance

<sup>1</sup> Source - National Academy of Science, 1980; *Drinking Water and Health - Volume 3*; Washington, D.C.

## SECTION 2

### EMISSIONS FROM AGRICULTURAL TILLING

The emission of soil particles into air during tilling operations depends mainly on the silt content of the soil (defined as percentage of particles less than 75 micrometers [ $\mu\text{m}$ ] in diameter). Because tilling and related operations are usually done only when the soil is reasonably dry, surface moisture content is generally not a key factor. Also, emissions do not depend heavily on the specific tillage implement, if operations are at a normal speed (usually 8 to 10 km/hr). Based on direct measurements, the emissions of soil per unit area during tilling of land is given by (USEPA, 1988f):

Equation No.

$$E = 5.38 k S^{0.6} \quad (1)$$

where:

E	=	Emission rate (kg/hectare)
k	=	Particle size multiplier (percent of total emissions below a specific size limit)
S	=	Silt content of surface soil (percent)

The value of k for particles less than 10  $\mu\text{m}$  (i.e., PM10) is 0.21 (USEPA, 1988f). The silt content in the Spoils Disposal Area is 62 percent. The value of S is not known at the other BAAP sites, so a value of 18 percent is assumed (USEPA, 1988f). Based on these parameters, the PM10 emission rate for areas other than the Settling Pond/Spoils Disposal Area is:

Equation No.

$$\begin{aligned} E &= (5.38)(0.21)(18)^{0.6} \\ &= 6.4 \text{ kg/hectare} \end{aligned} \quad (2)$$

Assuming the tractor is moving at 8 km/hr and is pulling an implement about 5 meters wide, it will take about 15 minutes to till one hectare (1 hectare = 10,000  $\text{m}^2$  = 2.5 acres). Based on this, the emission rate per unit area may be expressed as:

## APPENDIX M

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		<u>Equation No.</u>
E	= 6.4 kg/10,000 m <sup>2</sup> /15 min	(3)
	= 4.3E-5 kg/m <sup>2</sup> /min	
	= 7.1E-7 kg/m <sup>2</sup> /sec	

Calculation of PM10 Concentrations in Air. The concentration of PM10 in air resulting from farm tilling at each area was calculated using the box model (Hanna et al., 1982). The basic equation is:

		<u>Equation No.</u>
C	= $E \cdot X / (H/2 \cdot U)$	(4)

where:

C	=	Concentration of PM10 in air (kg/m <sup>3</sup> )
E	=	PM10 emission rate (kg/m <sup>2</sup> /sec)
X	=	Distance from upwind to downwind edge of the box (meters [m])
H	=	Mixing height of the box (m)
U	=	Windspeed across the box (m/sec)

Values of these parameters were derived as follows:

E	=	The emission rate is 7.1E-7 kg/m <sup>2</sup> /sec, calculated as described.
X	=	The distance from the upwind to downwind edge of the box is assumed to be 200 m. This corresponds to a square field size of 10 acres (4E+4 m <sup>2</sup> ), which is equal to or larger than the contaminated areas at most sites.
H	=	The mixing height of the box is a function of the upwind to downwind size of the box and the turbulence of the air. Turbulence, in turn, is a function of the roughness of the terrain. The value of H at the upwind edge of the site is zero. At the downwind edge, the value of H was calculated from the equation (Pasquill, 1975):

Equation No.

$$X = 6.25 ZO [(H/ZO) \ln (H/ZO) - 1.58 (H/ZO) + 1.58] \quad (5)$$

where:

$$\begin{aligned} X &= \text{Upwind to downwind distance (m)} \\ ZO &= \text{Roughness height (m). Based on a roughness height of 1 cm (0.01 m) for a plowed field, the value of H at 200 m is 6.5 m (USEPA, 1985c). The average height over the whole box is then H/2 (3.3 m).} \\ U &= \text{The average wind speed was taken to be 5.3 m/sec, based on wind speed at Milwaukee, Wisconsin (USEPA, 1985c).} \end{aligned}$$

Employing these input parameters, the concentration of PM10 in air over a field during tilling operations is calculated as follows:

Equation No.

(6)

$$(7.1E-7 \text{ kg/m}^2/\text{sec}) \cdot 200 \text{ m}$$

$$\begin{aligned} C &= 3.3 \text{ m} \cdot 5.3 \text{ m/sec} \\ &= 8.1E-6 \text{ kg/m}^3 \end{aligned}$$

**SECTION 3**  
**CALCULATION OF PARTICULATE EMISSION FACTOR**

**BADGER ARMY AMMUNITION PLANT**

$$PEF (m^3/kg) = \frac{LS \times V \times DH \times 3600s/hr}{A} \times \frac{1000g/kg}{0.036 \times (1-G) \times (U_m/U_t)^3 \times F(x)}$$

where:

<u>Parameter</u>	<u>Definition (units)</u>	<u>Default</u>
PEF	particulate emission factor (m <sup>3</sup> /kg)	4.63 x 10 <sup>9</sup> m <sup>3</sup> /kg
LS	width of contaminated area (m)	45 m
V	wind speed in mixing zone (m/s)	2.25 m/s
DH	diffusion height (m)	2 m
A	area of contamination (m <sup>2</sup> )	2,025 m <sup>2</sup>
0.036	respirable fraction (g/m <sup>2</sup> -hr)	0.036 g/m <sup>2</sup> -hr
G	fraction of vegetative cover (unitless)	0
U <sub>m</sub>	mean annual wind speed (m/s)	4.5 m/s
U <sub>t</sub>	equivalent threshold value of wind speed at 10 m (m/s)	12.8 m/s
F(x)	function dependent on U <sub>m</sub> /U <sub>t</sub> (unitless)	0.0497 (determined using USEPA 1985C)

## SECTION 4

### CALCULATION OF SOIL-TO-AIR VOLATILIZATION FACTOR (VF)<sup>1</sup> BADGER ARMY AMMUNITION PLANT

$$VF (m^3/kg) = \frac{(LS \times V \times MH)}{A} \times \frac{(3.14 \times ER \times T)^{0.5}}{(2 \times Dei \times E \times Kas \times CF)}$$

Where:

$$ER (cm^2/s) = \frac{(Dei \times E)}{E + (Ps)(1-E)/Kas}$$

and

$$Kas (g \text{ soil}/cm^3) = \frac{H \times 41}{Kd} \quad Dei = Di \times E^{0.33} \quad Kd = Koc \times OC$$

where:

- LS = width of contaminated area (m)
- V = wind speed in mixing zone (m/s)
- MH = mixing height (m)
- A = area of contamination (cm<sup>2</sup>)
- Dei = effective diffusivity (cm<sup>2</sup>/s)
- E = soil porosity (unitless)
- Kas = soil/air partition coefficient (g soil/cm<sup>3</sup> air)
- Ps = true soil density (g/cm<sup>3</sup>)
- T = exposure interval (s)
- OC = organic carbon content of soil (fraction)
- Di = molecular diffusivity (cm<sup>2</sup>/s)
- H = Henry's law constant (atm-m<sup>3</sup>/mol)
- Kd = soil-water partition coefficient (g/cm<sup>3</sup>)
- Koc = Organic carbon partition coefficient (cm<sup>3</sup>/g)
- CF = conversion factor (kg/g)

PARAMETER	VALUE	UNITS	SOURCE
LS	31	m	assumed
V	2.25	m/s	USEPA,1991a
MH	2	m	USEPA,1991a
A	1.10E+07	cm <sup>2</sup>	assumed
E	0.35		assumed
Ps	2.65	g/cm <sup>3</sup>	USEPA,1991a
T	7.90E+08	s	assumed
OC	0.02		USEPA,1991a
CF	0.001	kg/g	

# SECTION 4

## CALCULATION OF SOIL-TO-AIR VOLATILIZATION FACTOR (VF)<sup>1</sup> BADGER ARMY AMMUNITION PLANT

COMPOUND	Di (cm <sup>2</sup> /sec)	H (atm-m <sup>3</sup> / mol)	Kd (g/cm <sup>3</sup> )	Kas (g soil/ cm <sup>3</sup> air)	ER (cm <sup>2</sup> /s)	VF (m <sup>3</sup> /kg)
Benzene	9.23E-02	5.59E-03	1.66E+00	1.38E-01	1.78E-03	4.22E+03
Toluene	8.30E-02	6.37E-03	6.00E+00	4.35E-02	5.15E-04	8.01E+03
Acetone	1.24E-01	2.50E-05	1.10E+02	9.32E-06	1.66E-07	4.50E+05

	Koc
Benzene	83
Toluene	300
Acetone	2.2

<sup>1</sup> - Calculated for compounds where H > 1E-05 atm-m<sup>3</sup>/mol and molecular weight > 200 g/mole



# SECTION 5

## CALCULATION OF PROTECTIVE CONCENTRATIONS FOR INGESTION OF DRINKING WATER TARGET RISK SET AT 10<sup>-6</sup>

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### BADGER ARMY AMMUNITION PLANT

#### EXPOSURE PARAMETERS

#### EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
Concentration Water	CW	see below	mg/liter	Calculated
Ingestion Rate	IR	2	liters/day	USEPA, 1991b
Target Risk Level	TR	1x10 <sup>-6</sup>		USEPA, 1991a
Target Hazard Index	THI	1		USEPA, 1991a
Body Weight	BW	70	kg	USEPA, 1989d
Exposure Duration	ED	30	years	USEPA, 1991b
Exposure Frequency	EF	350	days/year	USEPA, 1991b
Averaging Time	AT	70	years	USEPA, 1989d
Cancer	AT	30	years	USEPA, 1991b
Noncancer				

$$CW \text{ (carcinogenic)} = \frac{TR \pm BW \pm AT \pm 365 \text{ days/year}}{\text{CANCER SLOPE FACTOR} \pm ED \pm EF \pm IR}$$

$$CW \text{ (noncarcinogenic)} = \frac{THI \pm BW \pm AT \pm 365 \text{ days/year}}{1/\text{REFERENCE DOSE} \pm ED \pm EF \pm IR}$$

USEPA, 1999d. Risk Assessment Guidance for Superfund, Part A  
USEPA, 1991a. Risk Assessment Guidance for Superfund, Part B  
USEPA, 1991b. Standard Default Exposure Factors

SECTION 3, continued  
 CALCULATION OF PROTECTIVE CONCENTRATIONS FOR INGESTION OF DRINKING WATER  
 TARGET RISK SET AT 10<sup>-6</sup>

BADGER ARMY AMMUNITION PLANT

PRELIMINARY REMEDIATION GOAL

COMPOUND	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	REFERENCE DOSE (mg/kg-day)	WATER CONCENTRATION - CARCINOGENIC (mg/L)	WATER CONCENTRATION - NONCARCINOGENIC (mg/L)
V	NA	7E-03		0.26
ZN	NA	2E-01		7.30
MNDPA	4.9E-03	ND	0.02	
BE	4.3E+00	5E-03	0.00002	0.18

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REMEDIAL INVESTIGATION  
BADGER ARMY AMMUNITION PLANT

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**APPENDIX N**  
**IRIS FILES FOR COMPOUNDS OF POTENTIAL CONCERN**

Option? CAS/71-55-6

File: 5 Count: 1

Option? TYPE 5/2/1

File: 5 Entry: 1

IRIS Accession Number 1197

(CAS) CAS Registry Number: 71-55-6  
(MAT) Material Name: 1,1,1-Trichloroethane  
(SYN) Synonyms: AEROTHENE TT;  
CHLOROETENE;  
CHLOROETHENE;  
CHLOROETHENE NU;  
CHLOROFORM, METHYL-;  
CHLOROTHANE NU;  
CHLOROTHENE;  
CHLOROTHENE NU;  
CHLOROTHENE VG;  
CHLORTEN;  
ETHANE, 1,1,1-TRICHLORO-;  
INHIBISOL;  
METHYLCHLOROFORM;  
METHYLTRICHLOROMETHANE;  
NCI-C04626;  
RCRA WASTE NUMBER U226;  
STROBANE;  
alpha-T;  
1,1,1-TCE;  
1,1,1-TRICHLOROETHANE;  
1,1,1-TRICHLOROETHANE;  
Trichloroethane, 1,1,1-;  
alpha-TRICHLOROETHANE;  
1,1,1-TRICHLOROETHANE;  
TRI-ETHANE;  
UN 2831

(UPD) Update Date: 01-01-92

(EFF) Effective Date: 01-01-92

(STAT) Status:

STATUS OF DATA FOR 1,1,1-Trichloroethane

File On-Line 03-31-87

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	withdrawn	08-01-91
Inhalation RfC Assessment (I.B.)	pending	

Carcinogenicity Assessment (II.)	on-line	09-01-90
Drinking Water Health Advisories (III.A.)	on-line	09-01-90
U.S. EPA Regulatory Actions (IV.)	on-line	01-01-92
Supplementary Data (V.)	no data	

=====

(HAZ) Chronic Health Hazards, Noncarcinogenic:

(HAZO) Hazards Oral:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

The oral RfD for this substance has been withdrawn pending further review by the RfD/RfC Work Group.

Contact: Michael L. Dourson / ORD / FTS/684-7544 or 513/569-7544

(HAZI) Hazards Inhalation:

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

A risk assessment for this substance/agent is under review by an EPA work group.

(CAR) Carcinogenicity Assessment:

(CARW) Carcinogenicity Weight:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity.

Basis -- There are no reported human data and animal studies (one lifetime gavage, one intermediate-term inhalation) have not demonstrated carcinogenicity. Technical grade 1,1,1-trichloroethane has been shown to be

weakly mutagenic, although the contaminant, 1,4-dioxane, a known animal carcinogen, may be responsible for this response.

II.A.2. HUMAN CARCINOGENICITY DATA

None.

II.A.3. ANIMAL CARCINOGENICITY DATA

Inadequate. The NCI (1977) treated Osborne-Mendel rats (50/sex/dose) with 750 or 1500 mg/kg technical-grade 1,1,1-trichloroethane 5 times/week for 78 weeks by gavage. The rats were observed for an additional 32 weeks. Twenty rats of each sex served as untreated controls. Low survival of both male and female treated rats (3%) may have precluded detection of a significant number of tumors late in life. Although a variety of neoplasms was observed in both treated and matched control rats, they were common to aged rats and were not dose-related. Similar results were obtained when the NCI (1977) treated B6C3F1 hybrid mice with the time-weighted average doses of 2807 or 5615 mg/kg 1,1,1-trichloroethane by gavage 5 days/week for 78 weeks. The mice were observed for an additional 12 weeks. The control and treated groups had 20 and 50 animals of each sex, respectively. Only 25 to 45% of those treated survived until the time of terminal sacrifice. A variety of neoplasms were observed in treated groups, but the incidence not statistically different from matched controls.

Quast et al. (1978) exposed 96 Sprague-Dawley rats of both sexes to 875 or 1750 ppm 1,1,1-trichloroethane vapor for 6 hours/day, 5 days/week for 12 months, followed by an additional 19-month observation period. The only significant sign of toxicity was an increased incidence of focal hepatocellular alterations in female rats at the highest dosage. It was not evident that a maximum tolerated dose (MTD) was used nor was a range-finding study conducted. No significant dose-related neoplasms were reported, but these dose levels were below those used in the NCI study.

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Mutagenicity testing of 1,1,1-trichloroethane has produced positive results in *S. typhimurium* strain TA100 (Simmon et al., 1977; Fishbein, 1979; Snow et al., 1979) as well as some negative results (Henschler et al., 1977; Taylor, 1978).

It was mutagenic for *S. typhimurium* strain TA1535 both with exogenous metabolic activation (Farber, 1977) and without activation (Nestmann et al., 1980). 1,1,1-Trichloroethane did not result in gene conversion or mitotic recombination in *Saccharomyces cerevisiae* (Farber, 1977; Simmon et al., 1977) nor was it positive in a host-mediated forward mutation assay using *Schizosaccharomyces pombe* in mice. The chemical also failed to produce chromosomal aberrations in the bone marrow of cats (Rampy et al., 1977), but

responded positively in a cell transformation test with rat embryo cells (Price et al., 1978).

An isomer, 1,1,2-trichloroethane, is carcinogenic in mice, inducing liver cancer and pheochromocytomas in both sexes. Dichloroethanes, tetrachloroethanes and hexachloroethanes also produced liver cancer in mice and other types of neoplasms in rats.

It should be noted that 1,4-dioxane, a known animal carcinogen that causes liver and nasal tumors in more than one strain of rats and hepatocellular carcinomas in mice, is a contaminant of technical-grade 1,1,1-trichloroethane.

(CARDOC) Carcinogenicity Documentation:

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

II.D.1. EPA DOCUMENTATION

U.S. EPA. 1984a. Health Effects Assessment for 1,1,1-Trichloroethane. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Emergency and Remedial Response, Washington, DC.

U.S. EPA. 1984b. Health Assessment Document for 1,1,1-Trichloroethane. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA-600/8-82-003F.

II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The 1984 Health Effects Assessment for 1,1,1-Trichloroethane has received limited Agency review. The values in the 1984 Health Assessment Document for 1,1,1-Trichloroethane have received both Agency and public review.

Agency Work Group Review: 08/05/87

Verification Date: 08/05/87

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Charlingayya Hiremath / ORD -- (202)260-5898 / FTS 260-5898

(HA) Hazard Assessment:  
(HAS) Health Advisories (Drinking Water):



### III. HEALTH HAZARD ASSESSMENTS FOR VARIED EXPOSURE DURATIONS

#### III.A. DRINKING WATER HEALTH ADVISORIES

##### III.A.1. ONE-DAY HEALTH ADVISORY FOR A CHILD

One-day HA --  $1 \times 10^2$  mg/L

NOAEL -- 1400 mg/kg/day

UF -- 100 (allows for interspecies and intrahuman variability with the use of

a NOAEL from an animal study)

Assumptions -- 1 L/day water consumption for a 10-kg child

Principal Study -- Vainio et al., 1976

A single oral dose of approximately 1400 mg/kg of 1,1,1-trichloroethane depressed some hepatic microsomal metabolic indices (including cytochrome P-450 and epoxide hydrazase) in rats but resulted in no other adverse effects. This level can be viewed as a NOAEL in this study.

##### III.A.2. TEN-DAY HEALTH ADVISORY FOR A CHILD

Appropriate data for calculating a Ten-day HA are not available. It is recommended that the Longer-term HA for the 10-Kg child of 40 mg/L be used as the Ten-day HA.

##### III.A.3. LONGER-TERM HEALTH ADVISORY FOR A CHILD

Longer-term (Child) HA --  $4 \times 10^1$  mg/L

NOAEL -- 350 mg/kg/day

UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)

Assumptions -- 1 L/day water consumption for a 10-kg child

Principal Study -- Bruckner et al., 1985

Rats were administered 1,1,1-trichloroethane by gavage 5 times/week for 12 weeks at levels of 0, 0.5, 2.5, or 5.0 g/kg/day. At levels above 0.5 g/kg reduced body weight gain and CNS effects were observed. Approximately 35% of these rats died during the first 50 days of the study. Also, the 5.0 g/kg/day dose group showed an increase in serum enzyme levels. The 0.5 g/kg/day level is identified as the NOAEL for this study. Based on a 7-day per week dosing regimen, this level would be equivalent to 350 mg/kg/day.

##### III.A.4. LONGER-TERM HEALTH ADVISORY FOR AN ADULT

Longer-term (Adult) HA --  $1E+2$  mg/L

NOAEL -- 350 mg/kg/day

UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)

Assumptions -- 2 L/day water consumption for a 70-kg adult

Principal Study -- Bruckner et al., 1985 (study described in III.A.3.)

#### III.A.5. DRINKING WATER EQUIVALENT LEVEL / LIFETIME HEALTH ADVISORY

DWEL --  $1E+0$  mg/L

Assumptions -- 20% exposure by drinking water

RfD Verification Date -- 05/15/86

Lifetime HA --  $2E-1$  mg/L

Principal Study -- McNutt et al., 1975

Male mice were continuously exposed to 1,1,1-trichloroethane via inhalation at 0, 1365 mg/cu.m, or 5460 mg/cu.m 6 hours/day for 14 weeks. Animals exposed to 5460 mg/cu.m displayed significant changes in the centrilobular hepatocytes. Based on the conditions of exposure and an assumed

absorption rate of 30%, the LOAEL of 1365 mg/cu.m is equivalent to 35 mg/kg/day.

#### III.A.6. ORGANOLEPTIC PROPERTIES

No information is available on the organoleptic properties of 1,1,1-trichloroethane.

#### III.A.7. ANALYTICAL METHODS FOR DETECTION IN DRINKING WATER

Analysis of 1,1,1-trichloroethane is by a purge-and-trap gas chromatographic procedure used for the determination of volatile organohalides in drinking water. Confirmatory analysis is by mass spectrometry.

#### III.A.8. WATER TREATMENT

Treatment technologies which will remove 1,1,1-trichloroethane from water include granular activated carbon adsorption and boiling. Air stripping is also an effective method; however, this process transfers the contaminant directly to the air stream.

#### III.A.9. DOCUMENTATION AND REVIEW OF HAS

U.S. EPA. 1985. Final Drinking Water Criteria Document on 1,1,1-Trichloroethane. Office of Drinking Water, Washington, DC.

EPA review of HAs in 1985.

Public reviews of HAs following notification of availability in October, 1985.

Science Advisory Board review of HAs in January, 1986.

Preparation date of this IRIS summary -- 08/20/90

### III.A.10. EPA CONTACTS

Charles Abernathy / ODW -- (202)260-5374 / FTS 260-5374

Edward V. Ohanian / ODW -- (202)260-7571 / FTS 260-7571

(REGS) Regulations:

(SDWA) Safe Drinking Water Act:

#### IV. U.S. EPA REGULATORY ACTIONS

##### IV.B. SAFE DRINKING WATER ACT (SDWA)

##### IV.B.1. MAXIMUM CONTAMINANT LEVEL GOAL (MCLG) for Drinking Water

Value (status) -- 200 ug/L (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- An MCLG of 200 ug/L for 1,1,1-trichloroethane is proposed based upon a DWEL and an assumed drinking water contribution of 20%. A DWEL of 1.0 mg/L was calculated based on liver toxicity in mice (inhalation study).

Reference -- 50 FR 46880 (11/13/85)

EPA Contact -- Health and Ecological Criteria Division / OST /  
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

##### IV.B.2. MAXIMUM CONTAMINANT LEVEL (MCL) for Drinking Water

Value (status) -- 200 ug/L (Final, 1987)

Considers technological or economic feasibility? -- NO

Discussion -- EPA has set an MCL equal to the MCLG.

Reference -- 52 FR 25690 (07/08/87); 56 FR 30266 (07/01/91)

Monitoring requirements -- All systems to be monitored for four consecutive quarters; repeat monitoring dependent upon detection, vulnerability status and system size.

Analytical methodology -- Gas chromatography (EPA 502.1, 502.2, 503.1);  
gas chromatographic/mass spectrometry (EPA 524.1, 524.2).

Best available technology -- Packed tower aeration; granular activated  
carbon.

EPA Contact -- Drinking Water Standards Division / OGWDW /  
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

(CWA) Clean Water Act:

IV.C. CLEAN WATER ACT (CWA)

IV.C.1. AMBIENT WATER QUALITY CRITERIA, Human Health

Water and Fish Consumption --  $1.84 \times 10^4$  ug/L

Fish Consumption Only --  $1.03 \times 10^0$  ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The WQC of  $1.84 \times 10^4$  ug/L is based on consumption of  
contaminated aquatic organisms and water. A WQC of 1.03 ug/L has also been  
established based on consumption of contaminated aquatic organisms alone.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS  
(202)260-1315 / FTS 260-1315

IV.C.2. AMBIENT WATER QUALITY CRITERIA, Aquatic Organisms

Freshwater:

Acute LEC --  $1.8 \times 10^4$  ug/L

Chronic LEC -- None

Marine:

Acute LEC --  $3.12 \times 10^4$  ug/L

Chronic LEC -- None

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but  
are the lowest effect levels found in the literature. LECs are given when the  
minimum data required to derive water quality criteria are not available.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS  
(202)260-1315 / FTS 260-1315

(FIFRA) Federal Insecticide, Fungicide, and Rodenticide Act:  
IV.D. FEDERAL INSECTICIDE, FUNGICIDE, AND RODENTICIDE ACT (FIFRA)  
IV.D.1. PESTICIDE ACTIVE INGREDIENT, Registration Standard

Status -- List "C" Pesticide

Reference -- 54 FR 30846 (07/24/89)

EPA Contact -- Registration Branch / OPP  
(703)557-7760 / FTS 557-7760

(TSCA) Toxic Substances Control Act:  
IV.E. TOXIC SUBSTANCES CONTROL ACT (TSCA)  
IV.E.1. TSCA, SECTION 6

Status -- Advance Notice of Proposed Rulemaking (ANPR) (1985)

Discussion -- EPA is developing a comprehensive and integrated strategy for a regulatory investigation of six solvents, including 1,1,1-trichloroethane.

Reference: 50 FR 42005 (10/17/85); 40 CFR 754

EPA Contact -- Chemical Control Division / OTS (202)260-3749 / FTS 260-3749

(RCRA) Resource Conservation and Recovery Act:  
IV.F. RESOURCE CONSERVATION AND RECOVERY ACT (RCRA)  
IV.F.1. RCRA APPENDIX IX, for Ground Water Monitoring

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)260-3000 / FTS 260-3000

(CERCLA) Superfund Act:  
IV.G. SUPERFUND (CERCLA)  
IV.G.1. REPORTABLE QUANTITY (RQ) for Release into the Environment

Value (status) -- 1000 pounds (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ is based on aquatic and chronic toxicity. Available data indicate a 96-hour Median Threshold Limit between 10 and 100 ppm, which corresponds to an RQ of 1000 pounds. RQ assignments based

on chronic toxicity reflect two primary attributes, the minimum effect dose (MED) levels for chronic exposure (mg/day for 70-kg man) and the type of effect (teratogenicity, etc.). The composite score of these attributes for this substance is 6.0, corresponding to an RQ of 1000 pounds.

Reference -- 50 FR 13456 (04/04/85); 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)260-3000 / FTS 260-3000

Option? LOGOFF

Your approximate total CIS session cost is \$ 15.61

CIS session terminated. CIS113491 logged off.  
Disconnected from 05AMS

Host Name: {NB^Z

Captured 6/11/92

1 - IRIS  
IRSN - 469  
DATE - 920604  
UPDT - 06/04/92, 52 fields  
STAT - Oral RfD Assessment (RDO) on-line 06/01/92  
STAT - Inhalation RfC Assessment (RDI) message 03/01/91  
STAT - Carcinogenicity Assessment (CAR) no data  
STAT - Drinking Water Health Advisories (DWHA) no data  
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92  
IRH - 03/01/91 RDI Inhalation RfC message on-line  
IRH - 03/01/91 REFS Bibliography on-line  
IRH - 09/01/91 RDO Oral RfD now under review  
IRH - 01/01/92 EXSR Regulatory Action section on-line  
IRH - 06/01/92 RDO Oral RfD summary on-line  
IRH - 06/01/92 OREF Oral RfD references added  
RLEN - 25212  
NAME - 2,4-Dinitrotoluene  
RN - 121-14-2  
SY - BENZENE, 1-METHYL-2,4-DINITRO-  
SY - 2,4-DINITROTOLUENE  
SY - 2,4-DINITROTOLUOL  
SY - 2,4-DNT  
SY - 1-METHYL-2,4-DINITROBENZENE  
SY - TOLUENE, 2,4-DINITRO-

RDO -

o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Neurotoxicity, Heinz bodies and biliary tract hyperplasia	NOAEL: 0.2 mg/kg/day LOAEL: 1.5 mg/kg/day	100	1 mg/kg/day	2E-3

Dog Feeding Study  
2-Year

Ellis et al., 1985

\*Conversion Factors: None

o ORAL RFD STUDIES :

Ellis, H.V., C.B. Hong, C.C. Lee, J.C. Dacre and J.P. Glennon. 1985.  
Subchronic and chronic toxicity studies of 2,4-dinitrotoluene. Part I. Beagle dogs. J. Am. College Toxicol. 4(4): 233-242.

Ellis et al. (1985) reported the results of a chronic toxicity study commissioned by the U.S. Army (Ellis et al., 1979) of dogs fed 98% pure 2,4-dinitrotoluene (2,4-DNT) for up to 24 months. Groups of beagle dogs (6/sex/dose) were fed 2,4-DNT in gelatin capsules at 0, 0.2, 1.5, or 10 mg/kg/day. In male dogs fed 10 mg/kg/day, 4 of the 6 males were sacrificed

moribund by study week 19 after exhibiting progressive paralysis. Neurotoxic effects, characterized by incoordination and paralysis, were exhibited by all dogs at this dose level within 6 months of study initiation and during month 16 in one dog receiving 1.5 mg/kg/day. CNS lesions included vacuolization, endothelial proliferation, and gliosis of the cerebellum. In dogs fed 1.5 and 10 mg/kg/day, there was methemoglobinemia with associated reticulocytosis and Heinz bodies; biliary tract hyperplasia; and pigmentation of the gallbladder, kidneys, and spleen. The hematologic effects were minimal during year 2, presumably due to an adaptive response. No males had testicular effects. The LOAEL in this study is 1.5 mg/kg/day based on neurotoxicity and the presence of Heinz bodies and biliary tract hyperplasia. The NOAEL is 0.2 mg/kg/day.

In a separate study (reported in Lee et al., 1978), groups of dogs (2/sex/dose) were given 2,4-DNT in capsules at doses of 0, 1, 5, or 25 mg/kg/day for 13 weeks. There was no apparent toxicity in the low- and mid-dose groups. In the high-dose group 2,4-DNT was toxic after 12-22 days and was lethal after 22 or more days. There was great variation in individual susceptibility. All affected dogs exhibited decreased food consumption, weight loss, urine stains on the fur, pale gums, neuromuscular incoordination, and paralysis. Hematological indices showed methemoglobinemia, anemia, and Heinz bodies. The dogs were in fair to poor nutritional condition with little or no body fat. Histologically, there was hemosiderosis in the liver and spleen, cloudy swelling of the kidneys in males and females, and aspermatogenesis in males. Dogs sacrificed during weeks 6 and 7 had brain lesions characterized by gliosis, edema, and demyelination of the cerebellum, spinal cord, and brain stem. After 4 weeks, dogs partially recovered from the various effects. The LOAEL is 25 mg/kg/day based on body weight loss, hematological abnormalities, neurological signs, and histopathology. The NOAEL is 5 mg/kg/day because no DNT-related effects were observed at this and lower doses.

Lee et al. (1985) reported the results of a chronic toxicity study commissioned by the U.S. Army (Ellis et al., 1979) of rats fed 98% pure 2,4-DNT in the diet for up to 24 months. Groups of CD (Sprague-Dawley) rats (38/sex) were provided an average 2,4-DNT intake of 0, 0.57, 3.9, or 34 mg/kg/day for males, and 0, 0.71, 5.1, or 45 mg/kg/day for females. After 12 months, 8 animals/sex/group were killed for necropsy; the remaining rats were sacrificed after 24 months. Four animals/sex/group were sacrificed at 13 and 25 months after being returned to normal diets for 1 month.

Cumulative deaths in high-dose males and females were significantly higher than in controls; 50% mortality occurred in high-dose rats by month 20 and in controls by month 23. Weight gains were reduced in high-dose animals (approximately 30-40%) and mid-dose (approximately 6-7%) animals compared with controls. Low-dose rats exhibited growth rates comparable to those of controls. Anemia and reticulocytosis occurred in mid- and high-dose males and in high-dose females after 12 months. The incidence of hyperplastic liver foci was increased in high-dose males (16/29) and mid-dose females (19/27). At 12 months, 6/7 high-dose males had marked atrophy of the testes with severe atrophy of the seminiferous tubules and almost complete lack of spermatogenesis. This lesion is common in geriatric rats, but is not normally seen in rats of this age. Beyond 12 months, severe atrophy of the seminiferous tubules occurred in 16% (4/25) of the controls, 26% (7/27) of the low-dose males, 33% (6/19) of the mid-dose males, and 81% (22/27) of the high-



dose males. The authors did not report the statistical significance of these effects. However, only the highest dose effect is significant by Chi square ( $p = 0.01$ ) and Fischer's Exact Test ( $p = 0.004$ ). The LOAEL is 34 mg/kg/day based on the incidence of changes in the seminiferous tubules of male rats. The NOAEL is 3.9 mg/kg/day.

In a separate study (Lee et al., 1978), groups of CD rats (16/sex/dose) were fed diets containing 0, 0.07, 0.20, or 0.7% 2,4-DNT (98% pure) for up to 13 weeks. The corresponding daily intakes were 0, 34, 93, or 266 mg/kg/day for males, and 0, 38, 108, or 145 mg/kg/day for females. Four animals/sex/group were sacrificed at 4 and 13 weeks after being returned to normal diets for 1 month. All high-dose females died within 3 weeks. One male in the mid-dose group and 6 in the high-dose group died between weeks 4 and 13. All surviving animals exhibited dose-dependent decreases in body weight gain, which ranged from approximately 9-55% when compared with controls. Food consumption was decreased in all dose groups. Orange to yellowish urine stains were observed on the fur of high-dose rats, and one male had widespread and stiff hind legs. Mid- and high-dose animals of both sexes were anemic, characterized by decreases in erythrocyte count, hematocrit, and hemoglobin, and concurrent reticulocytosis. Absolute liver and kidney weights were slightly increased in mid-dose males, and relative weights of these organs were significantly increased. There was splenic hemosiderosis in mid- and high-dose males and females. Spermatogenesis was decreased in mid-dose males and completely arrested in high-dose males. One high-dose male showed some signs of neuromuscular effects with demyelination in the cerebellum and brain stem. The LOAEL was 34 mg/kg/day based on decreased body weight gain and food consumption in male rats. There was no NOAEL because effects occurred at all doses tested.

Hong et al. (1985) reported the results of a chronic toxicity study commissioned by the U.S. Army (Ellis et al., 1979) of mice fed 98% pure 2,4-dinitrotoluene (2,4-DNT) in the diet for up to 24 months. Groups of 38 male and 38 female CD-1 mice were administered 2,4-DNT in their diets at average doses of 0, 14, 95, or 898 mg/kg/day. Both sexes of the high-dose animals and the males of the mid-dose groups had decreased weight gain that was approximately 10-22% lower than that of controls. High-dose males and females exhibited toxic anemia, reticulocytosis, and significant ( $p < 0.05$ ) increases in spleen and liver weights. All treated mice had an increased dose-related pigment in many tissues and organs including the liver, spleen, lungs, and kidney. High-dose females demonstrated ovarian atrophy. Mid- and high-dose males exhibited testicular atrophy.

In a separate study (Lee et al., 1978), groups of 16 male and 16 female CD-1 mice were fed diets containing 0, 0.07, 0.20, or 0.7% 2,4-DNT (98% pure) for 13 weeks. The corresponding daily intakes were 0, 47, 137, or 413 mg/kg/day for males, and 0, 52, 147, or 468 mg/kg/day for females. Five mice died during the study. Compared with controls, treated males exhibited a dose-dependent decrease in body weight (3, 11, and 19% from low to high dose) and, in the high-dose group only, there was decreased food consumption. The high-dose group of both sexes were anemic (decreased erythrocyte count, hematocrit, and hemoglobin) with concurrent reticulocytosis, mild hepatocellular dysplasia, and Kupffer cell dysplasia. High- and mid-dose males had mild degeneration of the seminiferous tubules or testicular degeneration. After 4 weeks off treatment, mice recovered completely. The

LOAEL was 47 mg/kg/day, based on body weight loss in males. There was no NOAEL because effects occurred at all doses tested.

Groups of 10 male Sprague-Dawley rats were administered 2,4-DNT (purity not reported) in corn oil by oral gavage at 0, 60, 180, or 240 mg/kg/day for 5 days (Lane et al., 1985). Significant reductions in the mating index and a sharp decrease in sperm-positive and pregnant females were observed in the 240-mg/kg/day dose group. Because of this finding, statistical evaluation of the reproductive results was difficult. No dominant lethal effects, characterized by early fetal deaths, were observed. Dose levels at or below 180 mg/kg/day did not result in changes in fertility or fetal death.

Bloch et al. (1988) fed groups of 9-10 Sprague-Dawley rats 2,4-DNT (97% pure) at dietary levels of 0, 0.1, or 0.2% (0, 1000, or 2000 ppm, respectively; or 0, 100, or 200 mg/kg/day, respectively). Effects observed in the highest dose group included significant body weight reduction ( $p < 0.05$ ), significant increases in serum follicle stimulating hormone and luteinizing hormone ( $p < 0.05$ ), significantly reduced sperm count ( $p < 0.01$ ), disruption of spermatogenesis, and histological alterations or degeneration in Sertoli cells, spermatocytes, and spermatids. No significant effects were observed in the low-dose rats.

In a 3-generation study conducted by Ellis et al. (1979), groups of 10-24 Sprague-Dawley rats/sex were fed diets containing 0, 15, 100, or 700 ppm (approximately 0, 0.75, 5, or 35 mg/kg/day, respectively) 2,4-DNT (98% pure) for up to 6 months prior to mating. Each parental generation produced two sets of offspring (Fa and Fb litters). The study was terminated during the third generation after weaning of the second litter (Fb). The highest dose was associated with reduced parental body weight, reduced pup survival, reduced fertility in F1 animals, and slightly lower mean litter size and pup weight. At mid- and low-dose levels there were slight reductions in body weight for first and third generation pups; however, parental fertility and offspring viability were not affected. The LOAEL is 700 ppm, based on severe reductions in fertility. The NOAEL is 100 ppm.

Technical grade DNT (76% 2,4-DNT; 19% 2,6-DNT; 5% other isomers) was administered in corn oil by gavage to groups of 5-20 time-mated female Fischer 344 rats on gestation days 7-20 (Price et al., 1985). The doses were 0, 14, 35, 37.5, 75, 100, or 150 mg/kg/day. In the 150 mg/kg/day group there was 46% mortality and clinical signs of toxicity began on gestation day 11. Mortality for the other treatment groups was similar to that of the control group. Corrected body weight gain (minus gravid uterine weight) was significantly reduced in dams receiving 14, 100, or 150 mg/kg/day. Relative liver weight was increased significantly in the 75- and 100-mg/kg/day groups. Relative spleen weight was significantly increased at all doses except 14 mg/kg/day. There were no treatment-related effects on the number of corpora lutea, implantations, live and dead fetuses, litter size, sex ratio, fetal weight, crown rump length, placental weight, or incidences of malformations and variations. There was a statistically insignificant increase in the percent resorptions in the 150-mg/kg/day group, which was considered to be indicative of a compound-related effect. Developmental effects noted in the fetuses were reduced liver weight at 14 mg/kg/day, and increased spleen weight at 35 and 75 mg/kg/day.

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o ORAL RFD UNCERTAINTY :

UF -- This uncertainty factor includes a factor of 10 for interspecies variability and a factor of 10 for intraspecies variability.

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o ORAL RFD MODIFYING FACTOR :

MF -- None

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o ORAL RFD COMMENTS :

Reported human health effects from DNT exposure are from occupational exposure studies in which workers were exposed primarily by inhalation with some contribution assumed from dermal absorption and ingestion (Etnier, 1987; Turner, 1986; Turner et al., 1985; Woollen et al., 1985). Major effects from chronic exposure include methemoglobinemia, characterized by Heinz body formation and compensatory reticulocytosis; cyanosis; neurotoxicity; and possible excess mortality from ischemic heart disease and residual circulatory system effects. Neurotoxicity is characterized by vertigo, paresthesia, tremors, unconsciousness, and paralysis. Humans appear to metabolize DNT qualitatively similar to animals with rapid absorption and urinary excretion of metabolites.

Heinz body formation has been observed in humans, dogs, and rodents that were exposed to DNT. Heinz bodies are thought to consist of denatured hemoglobin, possibly sulfhemoglobin, that may form disulfide bonds with red blood cell membranes and thus lead to impaired ion transport resulting in hyperpermeability and hemolysis (Smith, 1986). Cat, mouse, dog, and human erythrocytes are thought to be particularly susceptible to Heinz body formation.

Monitoring and production data indicate that the occurrence of 2,6-DNT is usually found in the presence of 2,4-DNT with the latter more significant by volume. Subchronic (13 week) studies in dogs, rats, and mice indicate that 2,4- and 2,6-DNT systemic toxicity may be qualitatively and quantitatively similar. Oral dosing studies with technical grade DNT (tg-DNT; approximately 75% 2,4-DNT, 20% 2,6-DNT, and 5% other isomers) do not elucidate the relative contribution of the various isomers to toxic effects.

Dinitrotoluene isomers are metabolized initially by liver oxidation (Rickert et al., 1984). Some metabolites are conjugated with sulfate or glucuronate and subsequently excreted in the urine or bile. The bile metabolites are hydrolyzed and reduced further by intestinal microflora. The bacterial metabolites are reabsorbed from the gut into the systemic circulation, oxidized in the liver, and excreted either in the urine or the bile for additional reduction by intestinal bacteria. There are species qualitative and quantitative differences; however, typical urinary metabolites of orally administered 2,4-DNT[ring-14C] in female CD rats, CD-1 mice, New Zealand white rabbits, beagle dogs, and rhesus monkeys were the glucuronide conjugates of 2,4-dinitrobenzyl alcohol and 2-amino-4-nitrobenzyl alcohol. Smaller amounts of 2,4-diaminotoluene, 2,4-diaminobenzyl alcohol, 2-amino-4-nitrotoluene, 4-amino-2-nitrotoluene, and 2,4-dinitrobenzoic acid were also recovered from each species. Several studies demonstrated similar urinary metabolites in male rats and mice. Humans exposed occupationally (via

inhalation and assumed dermal routes) to tg-DNT excreted some of the same urinary metabolites demonstrated in animals (e.g., the unchanged parent compound, 2,4-dinitrobenzyl alcohol, 2,4-dinitrobenzyl alcohol glucuronide, and 2,4-dinitrobenzoic acid) (Levine et al., 1985; Turner, 1986; Turner et al., 1985; Woolen et al., 1985). Other 2,4-DNT metabolites detected in the workers include 2-amino-4-nitrobenzoic acid, 4-amino-2-nitrobenzoic acid, 2-acetylamino-4-nitrobenzoic acid, and 4-acetylamino-2-nitrobenzoic acid.

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o ORAL RFD CONFIDENCE :

Study -- High  
Data Base -- High  
RfD -- High

The toxic effects observed in the 2-year dog study are based on an adequate number of animals of both sexes. In addition, a variety of gross, histological, hematologic, and clinical endpoints were evaluated. These effects are consistent with those reported to occur in exposed humans. The data base is rated high to medium because there are numerous acute, subchronic, chronic, and lifetime studies in several mammalian species. However, developmental toxicity studies with 2,4-DNT are lacking. Several rodent strains have been tested, and both sexes have been tested in all species. Pharmacokinetics and toxic effects demonstrated in laboratory animal species are consistent with observations from human exposure studies. The ratings for both the study and the data base result in a high to medium level of confidence in the RfD.

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o ORAL RFD SOURCE DOCUMENT :

Source Document -- U.S. EPA, 1990

Other EPA Documentation -- None

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o REVIEW DATES : 07/16/91, 08/14/91  
o VERIFICATION DATE : 08/14/91  
o EPA CONTACTS :

Welford C. Roberts / OST -- (202)260-7589

Edward V. Ohanian / OST -- (202)260-7571

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RDI -

o INHALATION RFD SUMMARY :

The health effects data for 2,4-dinitrotoluene were reviewed by the U.S. EPA RfD/RfC Work Group and determined to be inadequate for derivation of an inhalation RfC. The verification status of this chemical is currently not verifiable. For additional information on health effects of this chemical, interested parties are referred to the EPA documentation listed below.

U.S. EPA. 1980. Ambient Water Quality Criteria for Dinitrotoluenes. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-045. NTIS PB 81-117566/AS.

U.S. EPA. 1986. Health and Environmental Effects Profile for Dinitrotoluenes. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC. ECAO-CIN-P183. (Final Draft)

U.S. EPA. 1989. Ambient Water Quality Criteria Document Addendum for Dinitrotoluenes. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. ECAO-CIN-643. (Draft)

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o REVIEW DATES : 12/20/90

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WQCHU-

Water and Fish Consumption:  $1.1\text{E}-1$  ug/L

Fish Consumption Only:  $9.1\text{E}+0$  ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. However, zero may not be attainable at this time, so the recommended criteria represents an E-6 estimated incremental increase of cancer risk over a lifetime.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS  
(202)260-1315 / FTS 260-1315

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WQCAQ-

Freshwater:

Acute --  $3.3\text{E}+2$  ug/L

Chronic --  $2.3\text{E}+2$  ug/L

Marine:

Acute --  $5.9\text{E}+2$  ug/L

Chronic LEC --  $3.7\text{E}+2$  ug/L

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum database consisting of acute and chronic tests on a variety of species. Requirements and methods are

covered in the reference. The values that are indicated as "LEC" are not criteria but are the lowest level effect levels found in the literature. LEC's are given when the minimum data required to derive water quality criteria are not available.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS  
(202)260-1315 / FTS 260-1315

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CERC -

Value (status) -- 10 pounds (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ for 2,4-dinitrotoluene is based on potential carcinogenicity. Available data indicate a hazard ranking of medium and a weight of evidence classification of group B2, which corresponds to an RQ of 10 pounds.

Reference -- 54 FR 33418 (08/14/89)  
EPA Contact -- RCRA Superfund Hotline  
(800) 424-9346 / (703) 920-9810 / FTS 260-3000

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RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (703) 920-9810 / FTS 260-3000

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TSCA -

No data available

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OREF - Bloch, E., B. Gondos, M. Gatz, S.K. Varma and B. Thyssen. 1988.  
Reproductive toxicity of 2,4-dinitrotoluene in the rat. Toxicol. Appl.  
Pharmacol. 95: 466-472.  
OREF - Ellis, H.V., J.H. Hagensen, J.R. Hodgson, et al. 1979. Mammalian

- toxicity of munitions compounds. Phase III: Effects of lifetime exposure. Part I: 2,4- Dinitrotoluene. Final Report No. 7. U.S. Army Medical Bioengineering Research and Development Laboratory, Fort Detrick, MD. Order No. ADA077692. Available from NTIS, Springfield, VA.
- OREF - Ellis, H.V., C.B. Hong, C.C. Lee, J.C. Dacre and J.P. Glennon. 1985. Subchronic and chronic toxicity studies of 2,4-dinitrotoluene. Part I: Beagle dogs. *J. Am. College Toxicol.* 4(4): 233-242.
- OREF - Etnier, E.L. 1987. Water quality criteria for 2,4-dinitrotoluene and 2,6- dinitrotoluene. Final Report. U.S. Army Medical Bioengineering Research and Development Laboratory, Fort Detrick, MD, Order No. ADA188713. Available from NTIS, Springfield, VA.
- OREF - Hong, C.B., H.V. Ellis, C.C. Lee, H. S. 12, J.C. Dacre and J.P. Glennon. 1985. Subchronic and chronic toxicity studies of 2,4-dinitrotoluene. Part III: CD-1 mice. *J. Am. College Toxicol.* 4(4): 257-269.
- OREF - Lane, R.W., G.S. Simon, R.W. Dougherty, J.L. Egle and J.F. Borzelleca. 1985. Reproductive toxicity and the lack of dominant lethal effects of 2,4- dinitrotoluene in the male rat. *Drug Chem. Toxicol.* 8(4): 265-280.
- OREF - Lee, C.C., H.V. Ellis, J.J. Kowalski, et al. 1978. Mammalian toxicity of munitions compounds. Phase II: Effects of multiple doses. Part II: 2,4- Dinitrotoluene. Progress Report No. 3. U.S. Army Medical Bioengineering Research and Development Laboratory, Fort Detrick, MD, Order No. ADA061715. Available from NTIS, Springfield, VA.
- OREF - Lee C.C., C.B. Hong, H.V. Ellis, J.C. Dacre and J.P. Glennon. 1985. Subchronic and chronic toxicity studies of 2,4-dinitrotoluene. Part II: CD Rats. *J. Am. College Toxicol.* 4(4): 243-256.
- OREF - Levine, R.J., M.J. Turner, Y.S. Crume, M.E. Dale, T.B. Starr and D.E. Rickert. 1985. Assessing exposure to dinitrotoluene using a biological monitor. *J. Occup. Med.* 27(9): 627-638.
- OREF - Price, C.J., R.W. Tyl, T.A. Marks, L.L. Paschke, T.A. Ledoux and J.R. Reel. 1985. Teratologic evaluation of dinitrotoluene in the Fischer 344 rat. *Fund. Appl. Toxicol.* 5: 948-961.
- OREF - Rickert, D.E., B.E. Butterworth and J.A. Popp. 1984. Dinitrotoluene: Acute toxicity, oncogenicity, genotoxicity, and metabolism. *CRC Crit. Rev. Toxicol.* 13(3): 217-234.
- OREF - Smith, R.P. 1986. Toxic responses of the blood. In: Casarett and Doull's Toxicology, The Basic Science of Poisons, 3rd ed., C.D. Klaassen, M.O. Amdur and J. Doull, Eds. Macmillan Publishing Company, New York, NY. pp. 223-244.
- OREF - Turner, M.J. 1986. Identification and quantification of urinary metabolites of dinitrotoluenes in occupationally exposed humans. *CIIT Activities.* 6(2): 1-6.
- OREF - Turner, M.J., R.J. Levine, D.D. Nystrom, Y.S. Crume and D.E. Rickert. 1985. Identification and quantification of urinary metabolites of dinitrotoluenes in occupationally exposed humans. *Toxicol. Appl. Pharmacol.* 80: 166-174.
- OREF - U.S. EPA. 1990. Health Advisory for 2,4- and 2,6-dinitrotoluene (DNT). Office of Water, Washington, DC.
- OREF - Woollen, B.H., M.G. Hall, R. Craig and G.T. Steel. 1985. Dinitrotoluene: An assessment of occupational absorption during the manufacture of blasting explosives. *Int. Arch. Occup. Environ. Health.* 55: 319-330.
- IREF - U.S. EPA. 1980. Ambient Water Quality Criteria for Dinitrotoluenes. Prepared by the Office of Health and Environmental Assessment,

Environmental Criteria and Assessment Office, Cincinnati, OH, for the  
Office of Water Regulations and Standards, Washington, DC. EPA  
440/5-80-045. NTIS PB 81- 117566/AS.

IREF - U.S. EPA. 1986. Health and Environmental Effects Profile for  
Dinitrotoluenes. Prepared by the Office of Health and Environmental  
Assessment, Environmental Criteria and Assessment Office, Cincinnati,  
OH for the Office of Solid Waste and Emergency Response, Washington,  
DC. ECAO-CIN- P183. (Final Draft)

IREF - U.S. EPA. 1989. Ambient Water Quality Criteria Document Addendum for  
Dinitrotoluenes. Prepared by the Office of Health and Environmental  
Assessment, Environmental Criteria and Assessment Office, Cincinnati,  
OH. ECAO-CIN-643. (Draft)

CREF - None

HAREF- None



Captured 4/17/92

2 - IRIS  
IRSN - 582  
DATE - 920122  
STAT - Oral RfD Assessment (RDO) no data  
STAT - Inhalation RfC Assessment (RDI) no data  
STAT - Carcinogenicity Assessment (CAR) on-line 09/01/90  
STAT - Drinking Water Health Advisories (DWHA) no data  
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92  
IRH - 09/01/90 CAR Carcinogen assessment on-line  
IRH - 09/01/90 REFS Bibliography on-line  
IRH - 08/01/91 CREF Citations clarified  
IRH - 08/01/91 RDO Oral RfD now under review  
IRH - 09/01/91 RDO Oral RfD will be isomer-specific  
IRH - 01/01/92 EXSR Regulatory Action section on-line

RLEN - ND

NAME - Dinitrotoluene mixture, 2,4-/2,6-

RN - (01/01/92)

SY - 121-14-2

SY - BENZENE, 1-METHYL-2,4-DINITRO-

SY - 2,4-DINITROTOLUENE

SY - 2,4-DINITROTOLUOL

SY - 2,4-DNT

SY - HSDB 1144

SY - HSDB 2931

SY - 1-METHYL-2,4-DINITROBENZENE

SY - NCI-C01865

SY - NSC 7194

SY - RCRA WASTE NUMBER U105

SY - RCRA WASTE NUMBER U106

SY - TOLUENE, 2,4-DINITRO-

SY - 606-20-2

SY - 2,6-DINITROTOLUENE

SY - 2,6-DNT

SY - BENZENE, 2-METHYL-1,3-DINITRO-

SY - 2-METHYL-1,3-DINITROBENZENE

SY - TOLUENE, 2,6-DINITRO-

CAREV-

o CLASSIFICATION : B2; probable human carcinogen

o BASIS FOR CLASSIFICATION : Based on multiple benign and malignant tumor types at multiple sites in both sexes of rats (2 strains) and malignant renal tumors in male mice. The classification is supported by evidence of mutagenicity.

o HUMAN CARCINOGENICITY DATA :

None.

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o ANIMAL CARCINOGENICITY DATA :

Sufficient. Ellis et al. (1979) tested 2,4-DNT (98% 2,4-DNT and 2% 2,6-

DNT) in a chronic oral study using Charles River CD (Sprague-Dawley) rats (38/sex/dose) and CD-1 Swiss mice (58/sex/dose) for 2 years. Rats and mice were fed dietary concentrations of 0, 15, 100, and 700 ppm and 0, 100, 700, and 5000 ppm, respectively. Mortality was high in all treatment groups; the control group survival rate at 2 years was only 40-45% in rats and 20-30% in mice. In rats the test chemical induced increased incidences of hepatocellular carcinomas in high-dose males (1/25, 2/28, 2/19, 6/30) and a statistically significant increase in the same tumor type in high-dose females (0/23, 0/35, 1/27, 19/35). The incidence of hepatocellular neoplastic nodules was not considered statistically significantly elevated in any of the rat treatment groups. A statistically significant increase in the incidence of benign mammary gland tumors was observed in high-dose female rats (8/23, 9/35, 16/27, 33/35). Most male mice in the high-dose group died before 12 months and were not included in the incidence. In male mice the incidence of kidney tumors (both benign and malignant) was significantly elevated in the mid-dose group (0/20, 4/21, 15/17 for control, low and medium dose groups). No evidence of treatment-related increases in tumor frequency was noted in female mice.

In a 2-year NCI study (1978), 2,4-DNT (greater than 95% purity) was administered in the diet of Fischer 344 rats (50/sex/dose) and B6C3F1 mice (50/sex/dose) at doses of 80 and 200 ppm (rats) and 80 and 400 ppm (mice). Controls consisted of 75 rats/sex and 50 mice/sex. Rats and mice were on test for 78 weeks followed by an additional observation period of 13 to 26 weeks. Survival was adequate in all groups, and a reduced body weight gain in high dose groups indicated that an MTD had been approached; this indicates that the study conditions were valid. Only benign tumors were noted. 2,4-DNT induced a statistically significant increase in fibromas of the skin and subcutaneous tissue in male rats (0/71, 7/49, 13/49) and fibroadenomas of the mammary gland in high-dose female rats (13/71, 12/49, 23/50). No statistically significant increase in incidence of tumors was noted in male or female mice.

A CIIT study (1982) treated F344 rats (130/sex/dose) with technical grade DNT (76% 2,4-DNT and 19% 2,6-DNT) at dietary concentrations of 0, 3.5, 10.0 and 35.0 mg/kg/day. All male and female rats in the high-dose group were sacrificed at 55 weeks because of significantly reduced survival. Histopathological studies were performed on sacrificed animals (20 rats/sex) with 100% incidence of hepatocellular carcinoma in male rats (20/20) and 55% incidence in females (11/20). Mid- and low-dose animals were kept on test for 104 weeks. The incidences of liver carcinoma in males at 104 weeks were 1/61 for the control group, 9/70 for the low-dose group, 22/23 for the mid-dose group, and 20/20 (at 55 weeks) for the high-dose group; the incidences in females at 104 weeks were 0/57 for the control group, 0/61 for low-dose group, 40/68 for mid-dose group and 11/20 (at 55 weeks) for the high-dose group. The incidence of neoplastic nodules in males was 9/61, 11/70, 16/23, and 5/20, and the incidence in females was 5/57, 12/61, 53/68, and 12/20, at 104 weeks for the control, low-, mid- and (at 55 weeks) for the high-dose groups, respectively. Cholangiocarcinomas, presumably derived from the bile duct epithelium, were also observed in three high-dose males at 55 weeks and two mid-dose males at 104 weeks.

Leonard et al. (1987) treated groups of 20 F344 male rats with either technical-grade DNT, 2,4-DNT, or 2,6-DNT in the diet for 1 year. There was an untreated control group of 20 rats. Technical DNT (76% 2,4-DNT, 19% 2,6-DNT)

(35 mg/kg/day) induced hepatocellular carcinomas in 47% (9/19) of the treated males. 2,6-DNT (99.9% purity) induced hepatocellular carcinomas in 100% (19/19) of the high-dose rats (14 mg/kg/day) and 85% (17/20) of the low-dose (7 mg/kg/day). No tumors were found in controls or rats exposed to 2,4-DNT (99.9 purity) at 27 mg/kg/day. Two low-dose males receiving 2,6-DNT and two males receiving technical DNT developed cholangiocarcinoma. Although the duration of these studies was limited to 1 year and the number of animals tested was small, the data suggest that the 2,6-isomer accounts for much of the carcinogenic activity observed in previous mixed-isomer DNT bioassays.

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o SUPPORTING DATA :

The mutagenicity of dinitrotoluenes has been tested in numerous systems. 2,4-DNT causes reverse and forward mutations in several strains of *Salmonella typhimurium* (Couch et al., 1981; Tokiwa et al., 1981). DNA repair, as measured by UDS, was shown to occur in an in vivo male F344 rat hepatocyte assay (Mirsalis and Butterworth, 1982), but negative results were obtained in in vitro assays in rat hepatocytes (Bermudez et al., 1979) and spermatocytes (Working and Butterworth, 1984). Although Lee et al. (1978) observed an increased frequency of chromosomal aberrations in CD rat lymphocyte and kidney cultures, Ellis et al. (1979) observed no increased frequency in CD rat and beagle dog bone marrow and kidney cultures.

In a series of in vivo tumor initiation-promotion tests, Leonard and coworkers (Popp and Leonard, 1983; Leonard et al., 1983, 1986) compared the development of hepatic foci by the 2,4- and 2,6-DNT isomers and technical DNT.

Both 2,6- and technical DNT showed comparable initiating activity in partially-hepatectomized male F344 rats. In a promotion experiment, male F344 rats were initiated with a single dose of diethylnitrosamine prior to feeding 27 mg/kg/day 2,4-DNT or 7 mg/kg/day 2,6-DNT for 12 weeks. Positive results were observed for both 2,4 and 2,6-DNT, with the 2,6-isomer yielding a stronger response. These findings suggest the 2,6-isomer may be a complete hepatocarcinogen and 2,4-DNT a promoter.

In a skin-painting study using SENCAR mice, 2,6-DNT and 2,4-DNT were given as initiators (1, 5, or 10 mg) followed by TPA application for 30 weeks. Increased incidence of squamous cell carcinoma (5%) was observed in the 2,6-DNT-treated mice, although these results were not statistically significant (Slaga et al., 1985). When given intraperitoneally at 10 mg/kg followed by weekly TPA applications, 2,6-DNT produced 10% incidence of carcinomas, which was not significantly greater than controls. In the lung tumor bioassay, neither 1200 mg/kg of 2,4- nor 4800 mg/kg of 2,6-DNT administered intraperitoneally 3 times a week for 8 weeks increased the incidence of lung tumors in male A/Jax mice (Slaga et al., 1985). Schut et al. (1982), Stoner et al. (1984) and Maronpot et al. (1983) also reported negative results for 2,4-DNT administered orally or ip in the lung tumor bioassay with A/Jax mice, but positive results were reported using female A/St mice (Maronpot et al., 1983).

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CARO -

- o CLASSIFICATION : B2; probable human carcinogen
- o BASIS FOR CLASSIFICATION : Based on multiple benign and malignant tumor types at multiple sites in both sexes of rats (2 strains) and malignant renal tumors in male mice. The classification is supported by evidence of mutagenicity.
- o ORAL SLOPE FACTOR :  $6.8E-1$  per (mg/kg)/day
- o DRINKING WATER UNIT RISK :  $1.9E-5$  per (ug/L)
- o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure
- o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
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E-4 (1 in 10,000)	5 ug/L
E-5 (1 in 100,000)	$5E-1$ ug/L
E-6 (1 in 1,000,000)	$5E-2$ ug/L

o ORAL DOSE-RESPONSE DATA :

Tumor Type -- liver: hepatocellular carcinomas, neoplastic nodules; mammary gland: adenomas, fibroadenomas, fibromas, adenocarcinomas/carcinomas  
Test Animals -- Rat/Sprague-Dawley, female  
Route -- oral, diet  
Reference -- Ellis et al., 1979

Admin- istered (ppm)	Dose ----- Human Equivalent (mg/kg/day)	Tumor Incidence
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0	0	11/23
15	0.129	12/35
100	0.927	17/27
700	7.557	34/35

o ADDITIONAL COMMENTS :

The tumor incidences could be combined for quantitative purposes because the report by Ellis et al. (1979) provided pathology data for the individual animals. Transformed doses reflect the measured weight of the rats for each treatment period (0.425 kg control and low dose, 0.410 kg medium dose, 0.325 kg high dose).

The U.S. Army (ORNL, 1987) has calculated a quantitative risk estimate for the 2,6-isomer based on Leonard et al. (1987).

The unit risk should not be used if the water concentration exceeds 500

ug/L, since above this concentration the slope factor may differ from that stated.

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o DISCUSSION OF CONFIDENCE :

Relatively few animals were observed for a period of time approximating the lifespan of the animals. A slope factor of  $3.9E-1$  per (mg/kg)/day, obtained from renal tumors in male CD-1 mice (Ellis, 1979), is supportive of the risk estimate.

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CARDR-

o CARCINOGENICITY SOURCE :

U.S. EPA. 1986. Health and Environmental Effects Profile for Dinitrotoluene.

Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste, Washington, DC.

U.S. EPA. 1987. Health Effects Assessment for 2,4- and 2,6-Dinitrotoluene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.

The values in the Health and Environmental Effects Profile for Dinitrotoluene have received extensive Agency review.

DOCUMENT

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o REVIEW DATES : 04/01/87, 04/22/87, 05/25/88, 11/09/88,  
11/30/88,  
o VERIFICATION DATE : 05/03/89  
o EPA CONTACTS :

Robert Beliles / ORD -- (202)260-5898 / FTS 260-5898

Arthur Chiu / ORD -- (202)260-6764 / FTS 260-6764

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WQCHU-

Water and Fish Consumption:  $1.1E-1$  ug/L

Fish Consumption Only:  $9.1E0$  ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. However, zero may not be obtainable at this time, so the recommended criteria represents an E-6 estimated incremental increase of cancer risk over a lifetime.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS  
(202)260-1315 / FTS 260-1315

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WQCAQ-

Freshwater:

Acute --  $3.3E+2$  ug/L  
Chronic --  $2.3E+2$  ug/L

Marine:

Acute --  $5.9E+2$  ug/L  
Chronic LEC --  $3.7E+2$  ug/L

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute and chronic tests on a variety of species. Requirements and methods are covered in the reference to the Federal Register.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS  
(202)260-1315 / FTS 260-1315

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CERC -

Value (status) -- 10 pounds (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ is based on potential carcinogenicity. Available data indicate a hazard ranking of medium and a weight of evidence classification of Group B2, which corresponds to an RQ of 10 pounds.

Reference -- 54 FR 5311 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)260-3000 / FTS 260-3000

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RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)260-3000 / FTS 260-3000

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TSCA -

No data available

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OREF - None

IREF - None

CREF - Bermudez, E., D. Tillery and B.E. Butterworth. 1979. The effect of 2,4-Diaminotoluene and isomers of dinitrotoluene on unscheduled DNA synthesis in primary rat hepatocytes. Environ. Mutagen. 1: 391-398.

CREF - CIIT (Chemical Industry Institute of Toxicology). 1982. 104-Week Chronic Toxicity Study in Rats: Dinitrotoluene. Final Report, Vol. 1 and 2. Docket No. 12362. Research Triangle Park, NC.

CREF - Couch, D.B., P.F. Allen and D.J. Abernathy. 1981. The mutagenicity of dinitrotoluenes in Salmonella typhimurium. Mutat. Res. 90: 373-383.

CREF - Ellis, H.V. III, J.H. Hagensen, J.R. Hodgson, J.L. Minor and C.B. Hong. 1979. Mammalian Toxicity of Munitions Compounds. Phase III. Effects of Lifetime Exposure. Part I. 2,4-Dinitrotoluene. Report Order No. AD-A077692. p. 281.

CREF - Lee, C.C., H.V. Ellis, III, J.J. Kowalski, et al. 1978. Mammalian toxicity of munitions compounds. Phase II. Effects of multiple doses. Part II. 2,4- Dinitrotoluene. Midwest Research Institute, Kansas City MO. NTIS AD A061715.

CREF - Leonard, T.B., O. Lyght and J.A. Popp. 1983. Dinitrotoluene structure-dependent initiation of hepatocytes in vivo. Carcinogenesis. 4(8): 1059- 1061.

CREF - Leonard, T.B., T. Adams and J.A. Popp. 1986. Dinitrotoluene isomer-specific enhancement of the expression of diethylnitrosamine-initiated hepatocyte foci. Carcinogenesis. 7(11): 1797-1803.

CREF - Leonard, T.B., M.E. Graichen and J.A. Popp. 1987. Dinitrotoluene isomer- specific hepatocarcinogenesis in F344 rats. J. Natl. Cancer

Inst. 79(6): 1313-1319.

- CREF - Maronpot, R.R., H.P. Witschi, L.H. Smith and J.L. McCoy. 1983. Recent experience with the strain A mouse pulmonary tumor bioassay model. In: Short-term Bioassays in the Analysis of Complex Environmental Mixtures III. p. 341- 349.
- CREF - Mirsalis, J.C. and B.E. Butterworth. 1982. Induction of unscheduled DNA synthesis in rat hepatocytes following in vivo treatment with dinitrotoluene. Carcinogenesis. 3(3): 241-245.
- CREF - NCI (National Cancer Institute). 1978. Bioassay of 2,4-dinitrotoluene for possible carcinogenicity. Technical Report Series No. 54. U.S. Dept. Health, Education and Welfare, Washington, DC.
- CREF - Oak Ridge National Laboratory. 1987. Water Quality Criteria for 2,4-Dinitrotoluene and 2,6-Dinitrotoluene. Final Report for U.S. Army Medical Research and Development Command. AD-ORNL-6312.
- CREF - Popp, J.A. and T.B. Leonard. 1983. Hepatocarcinogenicity of 2,6-dinitrotoluene (DNT). Proc. Am. Assoc. Cancer Res. 24: 91.
- CREF - Schut, H.A.J., T.R. Loeb and G.D. Stoner. 1982. Distribution, elimination and test for carcinogenicity of 2,4-dinitrotoluene in strain A mice. Toxicol. Appl. Pharmacol. 64: 213-220.
- CREF - Slaga, T.J., L.L. Triplett, L. H. Smith and H.P. Witschi. 1985. Carcinogenesis of nitrated toluenes and benzenes, skin and lung tumor assays in mice. Final Report. ORNL/TM-9645. Oak Ridge National Laboratory, Oak Ridge, TN.
- CREF - Stoner, G.D., E.A. Greisiger, H.A.J. Schut, M.A. Pereira, T.R. Loeb, J.E. Klaunig and D.G. Branstetter. 1984. A comparison of the lung adenoma response in strain A/J mice after intraperitoneal and oral administration of carcinogens. Toxicol. Appl. Pharmacol. 72: 313-323.
- CREF - Tokiwa, H., R. Nakagawa and Y. Ohnishi. 1981. Mutagenic assay of aromatic nitro compounds with Salmonella typhimurium. Mutat. Res. 91: 321-325.
- CREF - U.S. EPA. 1986. Health and Environmental Effects Profile for Dinitrotoluene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste, Washington, DC.
- CREF - U.S. EPA. 1987. Health Effects Assessment for 2,4- and 2,6-Dinitrotoluene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.
- CREF - Working, P.K. and B.E. Butterworth. 1984. An assay to detect chemically induced DNA repair in rat spermatocytes. Environ. Mutagen. 6: 273-286.
- HAREF- None



(CAS) CAS Registry Number: 83-32-9

(MAT) Material Name: Acenaphthene

(SYN) Synonyms:  
Acenaphthylene, 1,2-dihydro-;  
Acenaphthene;  
HSDB 2659;  
Naphthyleneethylene;  
NSC 7657;  
PERI-ETHYLENENAPHTHALENE;  
1,2-DIHYDROACENAPHTHYLENE;  
1,8-ETHYLENENAPHTHALENE

(UPD) Update Date: 11-01-90

(EFF) Effective Date: 10-01-91

(STAT) Status:  
STATUS OF DATA FOR Acenaphthene

File On-Line 11-01-90

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	11-01-90
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	no data	
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	no data	
Supplementary Data (V.)	no data	

(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Hepatotoxicity	NOAEL: 175 mg/kg/day	3000	1	6E-2 mg/kg/day

Mouse Oral Subchronic LOAEL: 350 mg/kg/day  
Study

U.S. EPA, 1989  
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\*Conversion Factors: None

#### I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

U.S. EPA. 1989. Mouse oral subchronic study with acenaphthene. Study conducted by Hazelton Laboratories, Inc., for the Office of Solid Waste, Washington, DC.

Four groups of CD-1 mice (20/sex/group) were gavaged daily with 0, 175, 350, or 700 mg/kg/day acenaphthene for 90 days. The toxicological evaluations of this study included body weight changes, food consumption, mortality, clinical pathological evaluations (including hematology and clinical chemistry), organ weights and histopathological evaluations of target organs. The results of this study indicated no treatment-related effects on survival, clinical signs, body weight changes, total food intake, and ophthalmological alterations. Liver weight changes accompanied by microscopic alterations (cellular hypertrophy) were noted in both mid- and high-dose animals and seemed to be dose-dependent. Additionally, high-dose males and mid- and high-dose females showed significant increases in cholesterol levels. Although increased liver weights, without accompanying microscopic alterations or increased cholesterol levels, were also observed at the low dose, this change was considered to be adaptive and was not considered adverse. The LOAEL is 350 mg/kg/day based on hepatotoxicity); the NOAEL is 175 mg/kg/day.

#### I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF = 3000. An uncertainty factor of 3000 reflects 10 each for inter- and intraspecies variability, 10 for the use of a subchronic study for chronic RfD derivation, and 3 for the lack of adequate data in a second species and reproductive/developmental data.

MF = 1.

#### I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

Reshetyuk et al. (1970) examined the comparative toxicity of acenaphthene and acenaphthylene with respect to naphthalene. On intraperitoneal administration in rats (species/number/sex unspecified), naphthalene was more toxic than acenaphthene and acenaphthylene. Two LD<sub>50</sub> values (0.6 and 1.7 g/kg) were reported, but it is unclear to which of the three chemicals these values belonged. Intraperitoneal and intratracheal administration of naphthalene, acenaphthene, and acenaphthylene produced monotypic effects in the form of vascular disorders, and degeneration in the internal organs and central nervous system. Inflammatory changes were also observed in the lungs; the degree was the same for all three substances. Splenic degeneration was noted among the unscheduled deaths in this study. Reshetyuk et al. (1970) concluded that chronic inhalation of acenaphthene and acenaphthylene had more pronounced toxic effects than naphthalene.

Gershbein (1975) exposed partially hepatectomized rats to 15 mg/kg acenaphthene in the diet for 7 days. The only parameters used to assess toxicity were body weight, absolute liver weight, and liver regeneration. Information on histopathologic alterations and food intake is needed to evaluate the adversity of decreased body weight gain and increased liver weight observed in this study. Increased liver regeneration was reported.

Because of its inherent deficiencies, this study is not considered adequate for RfD derivation.

Knobloch et al. (1969) administered 2 g/kg acenaphthene orally to rats and mice for 32 days. Weight loss and mild histopathological alterations in the liver and kidney were observed. It is unclear whether experimental controls were used.

#### I.A.5. CONFIDENCE IN THE ORAL RfD

Study: Low  
Data Base: Low  
RfD: Low

Confidence in the study is low, because the observed effects were adaptive and not considered adverse. Confidence in the data base is low because of the lack of supporting chronic toxicity and developmental/reproductive studies. Low confidence in the RfD follows.

#### I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation -- U.S. EPA, 1980

Agency Work Group Review: 11/15/89

Verification Date: 11/15/89

#### I.A.7. EPA CONTACTS (ORAL RfD)

Harlal Choudhury / ORD -- (513)569-7536 / FTS 684-7536

Kenneth A. Poirier / ORD -- (513)569-7462 / FTS 684-7462

(CAR) Carcinogenicity Assessment:

#### II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

This substance/agent has not been evaluated by the U.S. EPA for evidence of human carcinogenic potential.

(CAS) CAS Registry Number: 208-96-8

(MAT) Material Name: Acenaphthylene

(SYN) Synonyms:  
Acenaphthylene;  
Cyclopenta(de)naphthalene;  
HSDB 2661;  
NSC 59821

(UPD) Update Date: 01-01-91

(EFF) Effective Date: 10-01-91

(STAT) Status:  
STATUS OF DATA FOR Acenaphthylene

File On-Line 01-01-91

Category (section)	Status	Last Revised
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Oral RfD Assessment (I.A.)	pending	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	01-01-91
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	no data	
Supplementary Data (V.)	no data	

(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS  
I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

A risk assessment for this substance/agent is under review by an EPA work group.

(CAR) Carcinogenicity Assessment:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE  
II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY  
II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity

Basis -- Based on no human data and inadequate data from animal bioassays.

#### II.A.2. HUMAN CARCINOGENICITY DATA

None.

#### II.A.3. ANIMAL CARCINOGENICITY DATA

Inadequate. No tumors were observed in a lifetime study, when 0.25% acenaphthylene (purity not specified) was applied to the skin (dose, frequency and duration not stated) of mice (sex and strain not specified) (Cook, 1932). Survival was 65% at 6 months, and 35% at 1 year. It is not stated whether a control group was used. In the series of experiments, however, the dermal application of other polycyclic aromatic hydrocarbons did result in the formation of mouse skin tumors.

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Acenaphthylene (1 mM) yielded positive results in a *Salmonella typhimurium* forward mutation assay (Kaden et al., 1979) and was not positive in a *Salmonella typhimurium* TA98 and TA100 in the presence of hepatic homogenates (Bos et al., 1988).

#### II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

None.

#### II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

None.

#### II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

##### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1990. Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons (PAHs). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. ECAO-CIN-D010, September, 1990. (Final Draft)

##### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The 1990 Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons has received Agency and external review.  
Agency Work Group Review: 02/07/90

Verification Date: 02/07/90

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Rita S. Schoeny / ORD -- (513)569-7544 / FTS 684-7544

Robert E. McGaughy / ORD -- (202)260-5889 / FTS 260-5889

FILE 1/2

File 1; Entry 1; Accession No. 1128

(CAS) CAS Registry Number: 67-64-1

(MAT) Material Name: Acetone

(SYN) Synonyms:

ACETON;  
Acetone;  
DIMETHYLFORMALDEHYDE;  
DIMETHYLKETAL;  
DIMETHYL KETONE;  
KETONE, DIMETHYL;  
KETONE PROPANE;  
beta-KETOPROPANE;  
METHYL KETONE;  
PROPANONE;  
2-PROPANONE;  
PYROACETIC ACID;  
PYROACETIC ETHER;  
RCRA WASTE NUMBER U002;  
UN 1090

(UPD) Update Date: 12-01-90

(EFF) Effective Date: 07-01-91

(STAT) Status:

STATUS OF DATA FOR Acetone

File On-Line 03-31-87

Category (section)	Status	Last Revised
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Oral RfD Assessment (I.A.)	on-line	12-01-90
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	12-01-90
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	on-line	07-01-90
Supplementary Data (V.)	no data	

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(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Increased liver and kidney weights and nephrotoxicity	NOEL: 100 mg/kg/day LOAEL: 500 mg/kg/day	1000	1	1E-1 mg/kg/day

Rat Oral Subchronic Study

U.S. EPA, 1986

\*Conversion Factors: Actual dose tested

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

U.S. EPA. 1986. Ninety-day gavage study in albino rats using acetone. Office of Solid Waste, Washington, DC.

Acetone was administered by gavage for 90 days to groups of albino rats (30/sex/group) at 0, 100, 500, or 2500 mg/kg/day. Body weights, food consumption, clinical chemistry, hematology, and histopathologic parameters, as well as organ weights and organ-to-body weight ratios, were measured and analyzed. Animals were sacrificed after 30 or 90 days of exposure. No effects were seen at the 100 mg/kg/day dose level throughout the study. RBC parameters were significantly increased in the 2500-mg/kg/day group at 30 days (males only) and at 90 days in males and females. Statistical analysis of the absolute and relative organ weight data revealed significantly increased kidney weights for females in the 500- and 2500-mg/kg/day groups and increased kidney-to-body and brain weight ratios for males and females in the 2500-mg/kg/day groups. Liver weight and liver/body weight ratios were also increased in the 2500-mg/kg/day males and females. Histopathologic studies revealed a marked increase in severity in tubular degeneration of the kidneys and hyaline droplet accumulation with increasing doses. This accumulation was significant in the 500- and 2500-mg/kg/day males and the 2500 mg/kg/day females.



Based on the above findings, the NOEL for this study is 100 mg/kg/day and the LOAEL is 500 mg/kg/day based on increased liver and kidney weights and nephrotoxicity.

#### I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF = 1000. An uncertainty factor of 1000 is used; 100 for inter- and intraspecies extrapolation and 10 to extrapolate from subchronic to chronic exposure.

MF = 1.

#### I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

Limited human studies have shown that workers exposed to acetone vapors (600 to 2150 ppm) experienced transient eye and nose irritation. Animals exposed to acetone vapors at 45,134 mg/cu.m experienced slight, but not significant, decreases in organ and body weights.

#### I.A.5. CONFIDENCE IN THE ORAL RfD

Study: Medium  
Data Base: Low  
RfD: Low

Confidence in the principal study is rated medium, since a moderate number of animals/dose/sex and an extensive number of parameters were measured. The data base is rated low because a very limited number of studies are available and no pertinent supporting studies were located. The overall confidence rating for the RfD is low.

#### I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation -- None

Agency RfD Work Group Review: 12/18/85, 05/30/86

Verification Date: 05/30/86

#### I.A.7. EPA CONTACTS (ORAL RfD)

Harlal Choudhury / ORD -- (513)569-7536 / FTS 684-7536

(CAR) Carcinogenicity Assessment:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity

Basis -- Based on lack of data concerning carcinogenicity in humans or animals.

II.A.2. HUMAN CARCINOGENICITY DATA

None.

II.A.3. ANIMAL CARCINOGENICITY DATA

None.

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Acetone did not show mutagenic activity when tested in *Salmonella typhimurium* strains TA98 and TA100 or in *Schizosaccharomyces pombe* strain P1 either in the presence or absence of liver homogenates (McCann et al., 1975; Abbondandolo et al., 1980; Maron et al., 1981; Hallstrom et al., 1981) or in cell transformation systems (Freeman et al., 1973; Rhim et al., 1974; Quarles et al., 1979a,b). Furthermore, acetone gave negative results in assays for chromosomal aberrations and sister chromatid exchange (Norppa et al., 1981; Norppa, 1981; Tates and Kriek, 1981), DNA binding (Kubinski et al., 1981), point mutation in mouse lymphoma cells (Amacher et al., 1980), and transfection of *E. coli* CR63 cells (Vasavada and Padayatty, 1981). In one study, however, acetone was reported to produce chromosomal aberrations but not sister chromatid exchanges (Kawachi et al., 1980).

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

None.

## II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

None.

## II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1988. Updated Health Effects Assessment for Acetone. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.

### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The 1988 updated Health Effects Document for Acetone has received Agency review and is approved for publication.

Agency Work Group Review: 12/06/89

Verification Date: 12/06/89

### II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Charles Ris / ORD -- (202)382-5895 / FTS 382-5898

## (REGS) Regulations:

### III. HEALTH HAZARD ASSESSMENTS FOR VARIED EXPOSURE DURATIONS

### IV. U.S. EPA REGULATORY ACTIONS

#### IV.A. CLEAN AIR ACT (CAA)

No data available

### IV.F. RESOURCE CONSERVATION AND RECOVERY ACT (RCRA)

#### IV.F.1. RCRA APPENDIX IX, for Ground Water Monitoring

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

IV.G. SUPERFUND (CERCLA)

IV.G.1. REPORTABLE QUANTITY (RQ) for Release into the Environment

Value (status) -- 5000 pounds (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- The final adjusted RQ for acetone is 5000 pounds, based on the application of the secondary criterion of biodegradation to the primary criteria RQ of 1000 pounds, determined by ignitability. Available data indicate a flash point of -4F and a boiling point of 133F, which

to an RQ of 1000 pounds. The final RQ takes biodegradation into account, since acetone biodegrades when released into the environment. The biological oxygen demand for 5 days (BOD5) is 46-55%.

Reference -- 50 FR 13456 (04/04/85)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

Option? FRG/AMMONIA

File: 14 Count: 1

Option? TYPE 14/1

File 14; Entry 1; Accession No. 1422

(CAS) CAS Registry Number: 7664-41-7

(MAT) Material Name: Ammonia

Option? TYPE 14/2

File 14; Entry 1; Accession No. 1422

(CAS) CAS Registry Number: 7664-41-7

(MAT) Material Name: Ammonia

(SYN) Synonyms:

Ammonia;  
AM-FOL;  
AMMONIA GAS;  
Ammonia Solution, Strong;  
Ammoniac [French];  
Ammoniaca [Italian];  
Ammoniak [German];  
Amoniaco [Spanish];  
Amoniak [Polish];  
ANHYDROUS AMMONIA;  
Aromatic Ammonia, Vaporole;  
Caswell No. 041;  
EPA Pesticide Chemical Code 005302;  
HSDB 162;  
Nitro-Sil;  
R 717;  
SPIRIT OF HARTSHORN;  
UN 1005;  
UN 2073;  
UN 2672

(UPD) Update Date: 05-01-91

(EFF) Effective Date: 07-01-91

(STAT) Status:  
STATUS OF DATA FOR Ammonia

File On-Line 05-01-91

Category (section) -----	Status -----	Last Revised -----
Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	on-line	05-01-91
Carcinogenicity Assessment (II.)	no data	
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	no data	
Supplementary Data (V.)	on-line	05-01-91

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(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

I.B.1. INHALATION RfC SUMMARY

Critical Effect -----	Exposures* -----	UF -----	MF ---	RfC -----
Lack of evidence of	NOAEL: 6.4 mg/cu.m (9.2 ppm)	30	1	1E-1
decreased pulmonary	NOAEL(ADJ): 2.3 mg/cu.m			mg/cu.m
function or changes	NOAEL(HEC): 2.3 mg/cu.m			
in subjective				
syptomatology	LOAEL: None			
Occupational Study				
Holness et al., 1989				
Increased severity of	NOAEL: None			
rhinitis and pneumonia				
with respiratory	LOAEL: 17.4 mg/cu.m (25 ppm)			
lesions	LOAEL(ADJ): 17.4 mg/cu.m			
	LOAEL(HEC): 1.9 mg/cu.m			
Rat Subchronic				

## Inhalation Study

Broderson et al., 1976

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\*Conversion Factors: MW = 17.03

Holness et al., 1989: Assuming 25C and 760 mm Hg, NOAEL (mg/cu.m) = 9.2 ppm

$\times 17.03/24.45 = 6.4 \text{ mg/cu.m}$ . The NOAEL is based on an 8-hour TWA occupational exposure. MVho = 10 cu.m/day, MVh = 20 cu.m/day. NOAEL(ADJ)

$= 6.4 \text{ mg/cu.m} \times (\text{MVho/MVh}) \times 5 \text{ days/7 days} = 2.3 \text{ mg/cu.m}$ .

Broderson et al., 1976: Assuming 25C and 760 mm Hg, the LOAEL (mg/cu.m) = 25 ppm  $\times 17.03/24.45 = 17.4 \text{ mg/cu.m}$ . The LOAEL(HEC) was calculated for a gas:respiratory effect in the ExtraThoracic region. MVa = 0.14 cu.m/day,

MVh = 20 cu.m/day, Sa(ET) = 11.6 sq. cm., Sh(ET) = 177 sq. cm. RGDR(ET) =

$(\text{MVa/Sa}) / (\text{MVh/Sh}) = 0.1068$ . NOAEL(HEC) =  $17.4 \times \text{RGDR} = 1.9 \text{ mg/cu.m}$ .

### I.B.2. PRINCIPAL AND SUPPORTING STUDIES (INHALATION RfC)

Holness, D.L., J.T. Purdham and J.R. Nethercott. 1989. Acute and chronic respiratory effects of occupational exposure to ammonia. Am. Ind. Hyg. Assoc. J. 50: 646-650.

Broderson, J.R., J.R. Lindsey and J.E. Crawford. 1976. The role of environmental ammonia in respiratory mycoplasmosis of rats. Am. J. Pathol. 85: 115-130.

Holness et al. (1989) investigated production workers exposed to ammonia in a soda ash facility. All of the available 64 production workers were invited to participate and 82% agreed to be evaluated. The control group consisted of 31 other plant workers from stores and office areas of the plant without previous exposure to ammonia. The mean age of the workers was 38.9 years and duration of exposure was 12.2 years. Weight was the only statistically significant difference in demographics found after comparing height, weight, years worked, % smokers and pack-years smoked. The mean TWA ammonia exposures based on personal sampling over one work shift (average sample collection 8.4 hours) of the exposed and control groups were 9.2 ppm (6.4 mg/cu.m) and 0.3 ppm (0.21 mg/cu.m), respectively.

A questionnaire was administered to obtain information on exposure and work histories and to determine eye, skin and respiratory symptomatology



(based on the American Thoracic Society [ATS] questionnaire [Ferris, 1978]). Spirometry (FVC, FEV-1, FEF50 and FEF75) was performed according to ATS criteria at the beginning and end of each work shift on the first workday of the week (day 1) and the last workday of the week (day 2). Differences in reported symptoms and lung function between groups were evaluated using the actual values and with age, height and pack-years smoked as covariates in linear regression analysis. Baseline lung function results were expressed as percent of predicted values calculated from Crapo et al. (1981) for FVC and FEV-1 and from Lapp and Hyatt (1967) for FEF50 and FEF75.

No statistical difference in the prevalence of the reporting symptoms was evident between the exposed and control groups, although workers reported that exposure at the plant had aggravated specific symptoms including coughing,

wheezing, nasal complaints, eye irritation, throat discomfort and skin problems. The percentage of exposed workers reporting hay fever or familial

history of hay fever was significantly less than controls, suggesting possible self-selection of atopic individuals out of this work force. The atopic status of the worker and control groups was not determined by skin prick tests to common aeroallergens. Furthermore, the workers complained that their symptomatology was exacerbated even though there was no statistical difference between groups. Since the study was cross-sectional in design with a small population, it is possible that selection bias may have occurred.

Baseline lung functions (based on the best spirometry values obtained during the four testing sessions) were similar in the exposed and control groups. No changes in lung function were demonstrated over either work shift

(days 1 or 2) or over the workweek in the exposed group compared with controls. No relationship was demonstrated between chronic ammonia exposure

and baseline lung function changes either in terms of the level or duration of exposure, probably due to lack of adequate exposure data for categorizing exposures and thus precluding development of a meaningful index accounting for both level and length of exposure.

Based on the lack of subjective symptomatology and changes in spirometry, this study establishes a free-standing TWA NOAEL of 9.2 ppm (6.4 mg/cu.m). Adjustment for the TWA occupational scenario results in a NOAEL(HEC) of 2.3 mg/cu.m.

Broderson et al. (1976) exposed groups of F344 rats (6/sex/dose) continuously to 25, 50, 150 or 250 ppm ammonia (HEC = 1.9, 3.7, 11.2 or 18.6 mg/cu.m, respectively) for 7 days prior to inoculation with *Mycoplasma pulmonis* and from 28-42 days following *M. pulmonis* exposure. Each treatment group had a corresponding control group exposed only to background ammonia and inoculated with *M. pulmonis* in order to produce murine respiratory mycoplasmosis (MRM). The following parameters were used to assess toxicity: clinical observations and histopathological examination of nasal passages, middle ear, trachea, lungs, liver and kidneys. All levels of ammonia, whether produced naturally or derived from a purified source, significantly increased the severity of rhinitis, otitis media, tracheitis and pneumonia characteristic of *M. pulmonis*. Furthermore, there was a significant concentration response between observed respiratory lesions and increasing environmental ammonia concentration for gross and microscopic lesions. All lesions observed were characteristic of MRM. Gross bronchiectasis and/or pulmonary abscesses and the extent of gross atelectasis and consolidation was consistently more prevalent in exposed animals at all concentrations than in their corresponding controls. The severity of the microscopic lesions in the nasal passages, middle ears, tracheas and lungs was significantly greater in all exposed groups compared with controls. Increasing ammonia concentration was not associated with an increasing frequency of *M. pulmonis* isolations. Additionally, rats not exposed to *M. pulmonis* and exposed to ammonia at 250 ppm developed nasal lesions (epithelial thickening and epithelial hyperplasia) unlike those observed in inoculated rats. Based upon these data in *M. pulmonis* exposed rats, a LOAEL(HEC) of 1.9 mg/cu.m was identified.

A group of 295 pathogen free F344 rats was inoculated with *M. pulmonis* and exposed to either trace or 100 ppm ammonia (HEC=7.4 mg/cu.m) (Schoeb et al., 1982). Growth of *M. pulmonis* was greater in exposed rats than in controls.

Similarly, serum immunoglobulin antibody responses to the inoculum were greater in the exposed population. It was further demonstrated that the nasal passages absorbed virtually all the ammonia at concentrations <500 ppm, indicating that the increased numbers of *M. pulmonis* in the lungs and the consequent exacerbation of lung lesions in MRM are secondary to events in the nasal passages rather than a direct effect of ammonia in the lung itself. These results are consistent with those of Broderson et al. (1976) detailed

above.

The use of Holness et al. (1989) as the principal study can only be supported in the context of the data array. It is not surprising that no effects were seen on screening spirometry since the exposure levels were low.

Comparing the 9.2 TWA of Holness et al. (1989) with other data on the respiratory effects of ammonia, a trend is observed that at lower concentrations the extrathoracic region of the respiratory system is affected

due to the chemical's solubility and reactivity; while at higher concentrations, the lower part of the respiratory system is involved in both

experimental animals (Dahlman, 1956; Gamble and Clough, 1976) and humans (Flury et al., 1983). Thus, no effects were observed in the lower respiratory system as reflected by pulmonary function. Pulmonary function may not be a

particularly sensitive test because exposure to this type of agent at low concentrations is not expected to result in significant exposure of the lower

respiratory region. No objective investigation of the workers' nasal epithelium was performed and the complaint of exacerbated upper respiratory

symptoms suggests sensory irritation and supports the extrathoracic region as

the critical region for an effect. The possibility of selection bias against

atopic predispositions in the population is suggested by the significantly

lower prevalence of hay fever in the exposed versus control cohort. Thus,

there is a concentration-response in the extrathoracic region in experimental

animals beginning at a LOAEL at essentially the same HEC as the NOAEL in Holness et al. (1989) and the NOAEL may be based on a less sensitive endpoint. Also the apparent discrepancy of a lower LOAEL(HEC) from Broderick et al. (1976) and the identified NOAEL(HEC) of the Holness et al. (1989) study may be the result of differences in air flow patterns since rats are obligate nose-

breathers and humans breathe oronasally. The use of the NOAEL from Holness et al. (1989) can be supported as marginal in this context due to the symptomatology complaints and because human data engenders less uncertainty

than extrapolation from the experimental animal data.

#### I.B.3. UNCERTAINTY AND MODIFYING FACTORS (INHALATION RFC)

UF - 30. An uncertainty factor of 10 is used to allow for the protection of sensitive individuals. A factor of 3 was used to account for several data base deficiencies including the lack of chronic data, the proximity of the

LOAEL to the NOAEL and the lack of reproductive and developmental toxicology studies. This factor is not larger than 3, however, since studies in rats (Schaerdel et al., 1983) have shown no increases in blood ammonia levels at exposures 32 ppm and only minimal increases at 300-1000 ppm, suggesting that no significant distribution is likely to occur at the HEC level calculated.

MF - 1.

#### I.B.4. ADDITIONAL STUDIES / COMMENTS (INHALATION RfC)

Groups of four healthy human volunteers were exposed weekly (5 days/week) to 25 (2 hours/day), 50 (4 hours/day) or 100 (6 hours/day) ppm ammonia (1.0, 4.1 or 12.1 mg/cu.m) for 6 weeks; or to 50 ppm (6.2 mg/cu.m) 6 hours/day for 6 weeks. Subjective and objective indications of eye and respiratory tract irritation, pulse rate, respiration rate, FVC, FEV and difficulty in performing simple cognitive tasks were used to assess toxicity. No abnormalities of the chest, heart, vital organs, neurological response, apparent motor function, or significant weight changes were observed during weekly medical examinations. Transient irritation of the nose and throat was observed at 50 ppm (duration-adjusted to 4.1 mg/cu.m) or greater (Ferguson et al., 1977).

#### I.B.5. CONFIDENCE IN THE INHALATION RfC

Study: Medium  
Data Base: Medium  
RfC: Medium

Confidence in the principal study is medium. Although a relatively small sample size (males only) was studied and a free standing NOAEL was determined, mild extrathoracic effects were observed in rats near the same HEC as reported in the Holness study. Additional human subchronic and acute studies support the NOAEL. Confidence in the data base is medium to high. Although developmental, reproductive or chronic toxicity following ammonia exposure has not been adequately tested, pharmacokinetic data suggests systemic distribution at the HEC level is unlikely. Reflecting medium confidence in the principal studies and medium to high confidence in the data base, confidence in the RfD is medium.

#### I.B.6. EPA DOCUMENTATION AND REVIEW OF THE INHALATION RfC

Source Document -- This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation -- U.S. EPA, 1987; U.S. EPA, 1989

Agency Work Group Review: 10/13/88, 09/19/89, 05/16/90, 09/19/90, 02/20/91

Verification Date: 02/20/91

I.B.7. EPA CONTACTS (INHALATION RfC)

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(CAR) Carcinogenicity Assessment:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

This substance/agent has not been evaluated by the U.S. EPA for evidence of human carcinogenic potential.

(PROP) Physical-Chemical Properties:

V.B. PHYSICAL-CHEMICAL PROPERTIES

Chemical Formula -- H3N

Molecular Weight -- 17.03

Boiling Point -- -28.03F, -33.35C (Merck, 1976)

Specific Gravity (H2O=1) -- Liquid 0.6818 at -33.35C (Merck, 1983; p. 74)

Vapor Pressure (mmHg) -- 400 at -45.4C (Weast, 1983)

Melting Point -- -107.9F, -77.7C (Merck, 1976)

Vapor Density (AIR=1) -- 0.6 (Weiss, 1980; p. 73)

Evaporation Rate (Butyl acetate=1) -- Not Found

Solubility in Water -- 31 g/100 g at 25C (Merck, 1983)

Appearance and Odor -- Colorless gas, liquid (Weast, 1979); sharp, cloying.

repellant odor (Booth, 1982)

Flash Point (Method Used) -- Not Found

**Flammable Limits:**

LEL -- 16% (NFPA, 1978)

UEL -- 25% (NFPA, 1978)

Conditions and Materials to Avoid -- Avoid mixing ammonia with other chemicals and water (Bretherick, 1979). Ammonia is incompatible with many materials including silver and gold salts, halogens, alkali metals, nitrogen trichloride, potassium chlorate, chromyl chloride, oxygen halides, acid vapors, azides, ethylene oxide (Bretherick, 1979), picric acid (Environment Canada, 1981), and many other chemicals (NFPA, 1978).

Hazardous Decomposition or Byproducts -- Not Found

Use -- Twenty-five percent of the ammonia produced is used as a direct application fertilizer; intermediate uses of ammonia include 10% used for urea fertilizer; 19% for ammonium nitrate fertilizer; 18% for all other fertilizers; 4% for ammonium nitrate-based commercial explosives; 7% for major fiber and plastic intermediates, and 14% for all other applications (SRI).

Ammonia is also used as a bactericide (USEPA/Pesticide Index, 1985).

Continue (Y/N/SKIP) (N)? N

Option? CAS/7440-38-2

File: 1 Count: 1

Option? TYPE 1/2/1

File: 1 Entry: 1

IRIS Accession Number 1278

(CAS) CAS Registry Number: 7440-38-2  
 (MAT) Material Name: Arsenic, inorganic  
 (SYN) Synonyms: Arsenic;  
 Arsenic, inorganic;  
 gray-arsenic  
 (UPD) Update Date: 01-01-92  
 (EFF) Effective Date: 01-01-92  
 (STAT) Status:  
 STATUS OF DATA FOR Arsenic, inorganic

File On-Line 02-10-88

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	10-01-91
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	02-01-91
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	on-line	01-01-92
Supplementary Data (V.)	no data	

(HAZ) Chronic Health Hazards, Noncarcinogenic:  
 (HAZO) Hazards Oral:  
 I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS  
 I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

NOTE: There was not a clear consensus among Agency scientists on the oral RfD. Applying the Agency's RfD methodology, strong scientific arguments can be made for various values within a factor of 2 or 3 of the currently

recommended RfD value, i.e., 0.1 to 0.8 ug/kg/day. It should be noted, however, that the RfD methodology, by definition, yields a number with inherent uncertainty spanning perhaps an order of magnitude. New data that possibly impact on the recommended RfD for arsenic will be evaluated by the Work Group as it becomes available. Risk managers should recognize the considerable flexibility afforded them in formulating regulatory decisions when uncertainty and lack of clear consensus are taken into account.

#### I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Hyperpigmentation, keratosis and possible vascular complications	NOAEL: 0.009 mg/L converted to 0.0008 mg/kg/day	3	1	3E-4 mg/kg/day
Human chronic oral exposure	LOAEL: 0.17 mg/L converted to 0.014 mg/kg/day			
Tseng, 1977; Tseng et al., 1968				

\*Conversion Factors: NOAEL was based on an arithmetic mean of 0.009 mg/L in a range of arsenic concentration of 0.001 to 0.017 mg/L. This NOAEL also included estimation of arsenic from food. Since experimental data were missing, arsenic concentrations in sweet potatoes and rice were estimated as 0.002 mg/day. Other assumptions included consumption of 4.5 L water/day and 55 kg bw (Abernathy et al., 1989).  $NOAEL = [(0.009 \text{ mg/L} \times 4.5 \text{ L/day}) + 0.002 \text{ mg/day}] / 55 \text{ kg} = 0.0008 \text{ mg/kg/day}$ . The LOAEL dose was estimated using the same assumptions as the NOAEL starting with an arithmetic mean water concentration from Tseng (1977) of 0.17 mg/L.  $LOAEL = [(0.17 \text{ mg/L} \times 4.5 \text{ L/day}) + 0.002 \text{ mg/day}] / 55 \text{ kg} = 0.014 \text{ mg/kg/day}$ .

#### I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Tseng, W.P. 1977. Effects and dose-response relationships of skin cancer and blackfoot disease with arsenic. Environ. Health Perspect. 19: 109-119.

Tseng, W.P., H.M. Chu, S.W. How, J.M. Fong, C.S. Lin and S. Yeh. 1968. Prevalence of skin cancer in an endemic area of chronic arsenicism in Taiwan. J. Natl. Cancer Inst. 40: 453-463.

The data reported in Tseng (1977) show an increased incidence of blackfoot



disease that increases with age and dose. Blackfoot disease is a significant adverse effect. The prevalences (males and females combined) at the low dose are 4.6 per 1000 for the 20-39 year group, 10.5 per 1000 for the 40-59 year group, and 20.3 per 1000 for the >60 year group. Moreover, the prevalence of blackfoot disease in each age group increases with increasing dose. However, a recent report indicates that it may not be strictly due to arsenic exposure (Lu, 1990). The data in Tseng et al. (1968) also show increased incidences of hyperpigmentation and keratosis with age. The overall prevalences of hyperpigmentation and keratosis in the exposed groups are 184 and 71 per 1000, respectively. The text states that the incidence increases with dose, but data for the individual doses are not shown. These data show that the skin lesions are the more sensitive endpoint. The low dose in the Tseng (1977) study is considered a LOAEL.

The control group described in Tseng et al. (1968; Table 3) shows no evidence of skin lesions and presumably blackfoot disease, although this latter point is not explicitly stated. This group is considered a NOAEL.

The arithmetic mean of the arsenic concentration in the wells used by the individuals in the NOAEL group is 9 ug/L (range: 1-17 ug/L) (Abernathy et al., 1989). The arithmetic mean of the arsenic concentration in the wells used by the individuals in the LOAEL group is 170 ug/L (Tseng, 1977; Figure 4). Using estimates provided by Abernathy et al. (1989), the NOAEL and LOAEL doses for both food and water are as follows: LOAEL -  $[170 \text{ ug/L} \times 4.5 \text{ L/day} + 2 \text{ ug/day (contribution of food)}] \times (1/55 \text{ kg}) = 14 \text{ ug/kg/day}$ ; NOAEL -  $[9 \text{ ug/L} \times 4.5 \text{ L/day} + 2 \text{ ug/day (contribution of food)}] \times (1/55 \text{ kg}) = 0.8 \text{ ug/kg/day}$ .

Although the control group contained 2552 individuals, only 957 (approximately 38%) were older than 20, and only 431 (approximately 17%) were older than 40. The incidence of skin lesions increases sharply in individuals above 20; the incidence of blackfoot disease increases sharply in individuals above 40 (Tseng, 1968; Figures 5, 6 and 7). This study is less powerful than it appears at first glance. However, it is certainly the most powerful study available on arsenic exposure to people.

This study shows an increase in skin lesions, 22% (64/296) at the high dose vs. 2.2% (7/318) at the low dose. The average arsenic concentration in the wells at the high dose is 410 ug/L and at the low dose is 5 ug/L (Cebrian et al., 1983; Figure 2 and Table 1) or 7 ug/L (cited in the abstract). The average water consumption is 3.5 L/day for males and 2.5 L/day for females. There were about an equal number of males and females in the study. For the dose estimates given below we therefore assume an average of 3 L/day. No data are given on the arsenic exposure from food or the body weight of the participants (we therefore assume 55 kg). The paper states that exposure times are directly related to chronological age in 75% of the cases. Approximately 35% of the participants in the study are more than 20 years old (Figure 1).

Exposure estimates (water only) are: high dose -  $410 \text{ ug/L} \times 3 \text{ L/day} \times (1/55 \text{ kg}) = 22 \text{ ug/kg/day}$ ; low dose -  $5\text{-}7 \text{ ug/L} \times 3 \text{ L/day} \times (1/55 \text{ kg}) = 0.3\text{-}0.4 \text{ ug/kg/day}$ .

The high-dose group shows a clear increase in skin lesions and is therefore designated a LOAEL. There is some question whether the low dose is a NOAEL or a LOAEL since there is no way of knowing what the incidence of skin lesions would be in a group where the exposure to arsenic is zero. The 2.2% incidence of skin lesions in the low-dose group is higher than that reported in the Tseng et al. (1968) control group, but the dose is lower (0.4 vs. 0.8 ug/kg/day).

The Southwick et al. (1983) study shows a marginally increased incidence of a variety of skin lesions (palmar and plantar keratosis, diffuse palmar or plantar hyperkeratosis, diffuse pigmentation, and arterial insufficiency) in the individuals exposed to arsenic. The incidences are 2.9% (3/105) in the control group and 6.3% (9/144) in the exposed group. There is a slight, but not statistically significant increase in the percent of exposed individuals that have abnormal nerve conduction (8/67 vs. 13/83, or 12% vs. 16% (Southwick et al., 1983; Table 8). The investigators excluded all individuals older than 47 from the nerve conduction portion of the study. These are the individuals most likely to have the longest exposure to arsenic.

Although neither the increased incidence of skin lesions nor the increase in abnormal nerve conduction is statistically significant, these effects may be biologically significant because the same abnormalities occur at higher doses in other studies. The number of subjects in this study was insufficient to establish statistical significance.

Table 3 (Southwick et al., 1983) shows the annual arsenic exposure from drinking water. No data are given on arsenic exposure from food or the body weight (assume 70 kg). Exposure times are not clearly defined, but are >5 years, and dose groups are ranges of exposure.

Exposure estimates (water only) are: dosed group -  $152.4 \text{ mg/year} \times 1 \text{ year}/365 \text{ days} \times (1/70) \text{ kg} = 6 \text{ ug/kg/day}$ ; control group -  $24.2 \text{ mg/year} \times 1 \text{ year}/365 \text{ days} \times (1/70) \text{ kg} = 0.9 \text{ ug/kg/day}$ .

Again because there are no data for a group not exposed to arsenic, there is some question if the control group is a NOAEL or a LOAEL. The incidence of skin lesions in this group is about the same as in the low-dose group from the Cebrian et al. (1983) study; the incidence of abnormal nerve conduction in the control group is higher than that from the low-dose group in the Hindmarsh et al. (1977) study described below. The control dose is comparable to the dose to the control group in the Tseng et al. (1968) and Hindmarsh et al. (1977) studies. The dosed group may or may not be a LOAEL, since it does not report statistically significant effects when compared to the control.

This study shows an increased incidence of abnormal clinical findings and abnormal electromyographic findings with increasing dose of arsenic (Hindmarsh et al., 1977; Tables III and VI). However, the sample size is extremely small. Percentages of abnormal clinical signs possibly attributed to As were 10, 16, and 40% at the low, mid and high doses, respectively. Abnormal EMG were 0, 17 and 53% in the same three groups.

The exact doses are not given in the Hindmarsh et al. (1977) paper; however, some well data are reported in Table V. The arithmetic mean of the arsenic concentration in the high-dose and mid-dose wells is 680 and 70 ug/L, respectively. Figure 1 (Hindmarsh et al., 1977) shows that the average arsenic concentration of the low-dose wells is about 25 ug/L. No data are given on arsenic exposure from food. We assume daily water consumption of 2

liters and body weight of 70 kg. Exposure times are not clearly stated.

Exposure estimates (water only) are: low -  $25 \text{ ug/L} \times 2 \text{ L/day} \times (1/70) \text{ kg}$   
 $= 0.7 \text{ ug/kg/day}$ ; mid -  $70 \text{ ug/L} \times 2 \text{ L/day} \times (1/70) \text{ kg} = 2 \text{ ug/kg/day}$ ; high -  $680$   
 $\text{ug/L} \times 2 \text{ L/day} \times (1/70) \text{ kg} = 19 \text{ ug/kg/day}$ .

The low dose is a no-effect level for abnormal EMG findings. However, because there is no information on the background incidence of abnormal clinical findings in a population with zero exposure to arsenic, there is no way of knowing if the low dose is a no-effect level or another marginal effect level for abnormal clinical findings. The low dose is comparable to the dose received by the control group in the Tseng (1977) and Southwick et al. (1983) studies.

The responses at the mid dose do not show a statistically significant increase but are part of a statistically significant trend and are biologically significant. This dose is an equivocal NOAEL/LOAEL. The high dose is a clear LOAEL for both responses.

As discussed previously there is no way of knowing whether the low doses in the Cebrian et al. (1983), Southwick et al. (1983) and Hindmarsh et al. (1977) studies are NOAELs for skin lesions and/or abnormal nerve conduction. However, because the next higher dose in the Southwick and Hindmarsh studies only shows marginal effects at doses 3-7 times higher, the Agency feels comfortable in assigning the low doses in these studies as NOAELs.

The Tseng (1977) and Tseng et al. (1968) studies are therefore considered superior for the purposes of developing an RfD and show a NOAEL for a sensitive endpoint. Even discounting the people <20 years of age, the control group consisted of 957 people that had a lengthy exposure to arsenic with no evidence of skin lesions.

The following is a summary of the defined doses in mg/kg/day from the principal and supporting studies:

- 1) Tseng (1977): NOAEL =  $8\text{E-}4$ ; LOAEL =  $1.4\text{E-}2$
- 2) Cebrian et al. (1983): NOAEL =  $4\text{E-}4$ ; LOAEL =  $2.2\text{E-}2$
- 3) Southwick et al. (1983): NOAEL =  $9\text{E-}4$ ; LOAEL = none (equivocal effects at  $6\text{E-}3$ )
- 4) Hindmarsh et al., 1977: NOAEL =  $7\text{E-}4$ ; LOAEL =  $1.9\text{E-}2$  (equivocal effects at  $2\text{E-}3$ )

#### I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF = 3. The UF of 3 is to account for both the lack of data to preclude reproductive toxicity as a critical effect and to account for some uncertainty in whether the NOAEL of the critical study accounts for all sensitive individuals.

MF = 1.

#### I.A.4. ADDITIONAL STUDIES / COMMENTS (ORAL RfD)

Ferm and Carpenter (1968) produced malformations in 15-day hamster fetuses via intravenous injections of sodium arsenate into pregnant dams on day 8 of gestation at dose levels of 15, 17.5, or 20 mg/kg bw. Exencephaly, encephaloceles, skeletal defects and genitourinary systems defects were produced. These and other terata were produced in mice and rats all at levels around 20 mg/kg bw. Minimal effects or no effects on fetal development have been observed in studies on chronic oral exposure of pregnant rats or mice to relatively low levels of arsenic via drinking water (Schroeder and Mitchner, 1971). Nadeenko et al. (1978) reported that intubation of rats with arsenic solution at a dose level of 25 ug/kg/day for a period of 7 months, including pregnancy, produced no significant embryotoxic effects and only infrequent slight expansion of ventricles of the cerebrum, renal pelvis and urinary bladder. Hood et al. (1977) reported that very high single oral doses of arsenate solutions (120 mg/kg) to pregnant mice were necessary to cause prenatal fetal toxicity, while multiple doses of 60 mg/kg on 3 days had little effect.

Extensive human pharmacokinetic, metabolic, enzymic and long-term information is known about arsenic and its metabolism. Valentine et al. (1987) established that human blood arsenic levels did not increase until daily water ingestion of arsenic exceeded approximately 250 ug/day (approximately 120 ug of arsenic/L. Methylated species of arsenic are successively 1 order of magnitude less toxic and less teratogenic. Some evidence suggests that inorganic arsenic is an essential nutrient in goats, chicks, mini pigs and rats. No comparable data are available for humans.

#### I.A.5. CONFIDENCE IN THE ORAL RfD

Study: Medium  
Data Base: Medium  
RfD: Medium

Confidence in the chosen study is considered medium. An extremely large number of people were included in the assessment (>40,000) but the doses were not well-characterized and other contaminants were present. The supporting human toxicity data base is extensive but somewhat flawed. Problems exist with all of the epidemiological studies. For example, the Tseng studies do not look at potential exposure from food or other source. A similar criticism can be made of the Cebrian et al. (1983) study. The U.S. studies are too small in number to resolve several issues. However, the data base does support the choice of NOAEL. It garners medium confidence. Medium confidence in the RfD follows.

#### I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- The only U.S. EPA documentation for this RfD is on IRIS.

Other EPA Documentation -- U.S. EPA, 1984, 1988

Source Document Review -- This analysis has been reviewed by EPA's Risk Assessment Council on 11/15/90.

This assessment was discussed by the Risk Assessment Council of EPA on 11/15/90 and verified through a series of meetings during the 1st, 2nd and 3rd quarters of FY91.

Agency Work Group Review: 03/24/88, 05/25/88, 03/21/89, 09/19/89, 08/22/90, 09/20/90

Verification Date: 11/15/90

#### I.A.7. EPA CONTACTS (ORAL RfD)

Charles Abernathy / OW -- (202)260-5374 / FTS 260-5374

Michael Dourson / ORD -- (513)569-7533 / FTS 684-7533

(CAR) Carcinogenicity Assessment:

(CARW) Carcinogenicity Weight:

#### II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

##### II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

##### II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- A; human carcinogen

Basis -- based on observation of increased lung cancer mortality in populations exposed primarily through inhalation and on increased skin cancer incidence in several populations consuming drinking water with high arsenic

concentrations.

#### II.A.2. HUMAN CARCINOGENICITY DATA

Studies of smelter worker populations (Tacoma, WA; Magma, UT; Anaconda, MT; Ronnskar, Sweden; Saganoseki-Machii, Japan) have all found an association between occupational arsenic exposure and lung cancer mortality (Enterline and Marsh, 1982; Lee-Feldstein, 1983; Axelson et al., 1978; Tokudome and Kuratsune, 1976; Rencher et al., 1977). Both proportionate mortality and cohort studies of pesticide manufacturing workers have shown an excess of lung cancer deaths among exposed persons (Ott et al., 1974; Mabuchi et al., 1979). One study of a population residing near a pesticide manufacturing plant revealed that these residents were also at an excess risk of lung cancer (Matanoski et al., 1981). Case reports of arsenical pesticide applicators have also demonstrated an association between arsenic exposure and lung cancer (Roth, 1958).

A cross-sectional study of 40,000 Taiwanese exposed to arsenic in drinking water found significant excess skin cancer prevalence by comparison to 7500 residents of Taiwan and Matsu who consumed relatively arsenic-free water (Tseng et al., 1968). This study design limited its usefulness in risk estimation. Arsenic-induced skin cancer has also been attributed to water supplies in Chile, Argentina and Mexico (Borgono and Greiber, 1972; Bergoglio, 1964; Cebrian et al., 1983). No excess skin cancer incidence has been observed in U.S. residents consuming relatively high levels of arsenic in drinking water (Morton et al., 1976; Southwick et al., 1981). The results of these U.S. studies, however, are not necessarily inconsistent with the existing findings from the foreign populations. The statistical powers of the U.S. studies are considered to be inadequate because of the small sample size.

A follow-up study (Tseng, 1977) of the population living in the same area of Taiwan, where arsenic contamination of the water supply was endemic, found significantly elevated standard mortality ratios for cancer of the bladder, lung, liver, kidney, skin and colon. This study of bladder, liver and lung cancer cases in the endemic area found a significant association with arsenic exposure that was dose-related. The association of arsenic ingestion and cancer of various internal organs has also been cited in a number of case reports (Chen et al., 1985, 1986). Persons treated with arsenic-containing medicinals have also been shown to be at a risk of skin cancer (Sommers and McManus, 1953).

#### II.A.3. ANIMAL CARCINOGENICITY DATA

None. There has not been consistent demonstration of arsenic carcinogenicity in test animals for various chemical forms administered by different routes to several species (IARC, 1980). There are some data to indicate that arsenic may produce animal tumors if retention time in the lung can be increased (Pershagen et al., 1982, 1984).

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Sodium arsenate has been shown to transform Syrian hamster embryo cells (Dipaolo and Casto, 1979) and to produce sister-chromatid-exchange in DON cells, CHO cells and human peripheral lymphocytes exposed in vitro (Wan et al., 1982; Ohno et al., 1982; Larramendy et al., 1981; Andersen, 1983; Crossen, 1983). While arsenic compounds have not been shown to mutate bacterial strains, it produces preferential killing of repair deficient strains (Rossman, 1981).

(CARO) Carcinogenicity Oral:

#### II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

The Risk Assessment Forum has completed a reassessment of the carcinogenicity risk associated with ingestion of inorganic arsenic. This report, which has been extensively peer-reviewed by outside reviewers (including SAB review) concluded that the most appropriate basis for an oral quantitative estimate was the study by Tseng et al. (1977), which reported increased prevalence of skin cancers in humans as a consequence of arsenic exposure in drinking water. Based on this study a unit risk of  $5E-5/\mu\text{g}/\text{L}$  was proposed.

A recent memorandum by the Administrator of the EPA recommended that the above unit risk be adopted. The memorandum further counsels that "in reaching risk management decisions in a specific situation, risk managers must recognize and consider the qualities and uncertainties of risk estimates. The uncertainties associated with ingested inorganic arsenic are such that estimates could be modified downwards as much as an order of magnitude, relative to risk estimates associated with most other carcinogens. In such instances, the management document must clearly articulate this fact and state the factors that influenced such a decision."

(CARI) Carcinogenicity Inhalation:

#### II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

##### II.C.1. SUMMARY OF RISK ESTIMATES



Inhalation Unit Risk --  $4.3\text{E-}3/\text{ug}/\text{cu.m}$

Extrapolation Method -- absolute-risk linear model

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
-----	-----
E-4 (1 in 10,000)	$2\text{E-}2 \text{ ug}/\text{cu.m}$
E-5 (1 in 100,000)	$2\text{E-}3 \text{ ug}/\text{cu.m}$
E-6 (1 in 1,000,000)	$2\text{E-}4 \text{ ug}/\text{cu.m}$

#### II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION EXPOSURE

Tumor Type -- lung cancer

Test Animals -- human, male

Route -- inhalation, occupational exposure

Reference -- Brown and Chu, 1983a,b,c; Lee-Feldstein, 1983; Higgins, 1982;

Enterline and Marsh, 1982

##### Ambient Unit Risk Estimates

Exposure		Unit	Geometric Mean	Final Estimates
Source	Study	Risk	Unit Risk	Unit Risk
Anaconda smelter	Brown and Chu,	$1.25 \text{ E-}3$		
	1983a,b,c			
	Lee-Feldstein, 1983	$2.80 \text{ E-}3$	$2.56 \text{ E-}3$	
	Higgins, 1982;	$4.90 \text{ E-}3$		$4.29 \text{ E-}3$
	Higgins et al., 1982;			
	Welch et al., 1982			
ASARCO smelter	Enterline and	$6.81 \text{ E-}3$	$7.19 \text{ E-}3$	
	Marsh, 1982	$7.60 \text{ E-}3$		

#### II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE)

A geometric mean was obtained for data sets obtained within distinct exposed populations (U.S. EPA, 1984). The final estimate is the geometric mean of those two values. It was assumed that the increase in age-specific mortality rate of lung cancer was a function only of cumulative exposures.

The unit risk should not be used if the air concentration exceeds  $2 \text{ ug}/\text{cu.m}$ , since above this concentration the unit risk may not be appropriate.

#### II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOSURE)

Overall a large study population was observed. Exposure assessments included air measurements for the Anaconda smelter and both air measurements and urinary arsenic for the ASARCO smelter. Observed lung cancer incidence was significantly increased over expected values. The range of the

estimates derived from data from two different exposure areas was within a factor of 6.

(CARDOC) Carcinogenicity Documentation:

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

II.D.1. EPA DOCUMENTATION

U.S. EPA. 1984. Health Assessment Document for Inorganic Arsenic. Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA 600/8-83-021F.

II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The 1984 Health Assessment Document for Inorganic Arsenic received Agency and external review including a review by SAB.

Agency Work Group Review: 01/13/88

Verification Date: 01/13/88

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

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Chao W. Chen / ORD -- (202)260-5898 / FTS 260-5898

(REGS) Regulations:

(CAA) Clean Air Act:

III. HEALTH HAZARD ASSESSMENTS FOR VARIED EXPOSURE DURATIONS

IV. U.S. EPA REGULATORY ACTIONS

IV.A. CLEAN AIR ACT (CAA)

No data available

(SDWA) Safe Drinking Water Act:

IV.B. SAFE DRINKING WATER ACT (SDWA)

IV.B.1. MAXIMUM CONTAMINANT LEVEL GOAL (MCLG) for Drinking Water

Value (status) -- 0.05 mg/L (Proposed, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- An MCLG of 0.05 mg/L for arsenic is proposed based on the current MCL of 0.05 mg/L. Even though arsenic is potentially carcinogenic in

humans by inhalation and ingestion, its potential essential nutrient value was

considered in determination of an MCLG. The basis for this evaluation is nutritional requirements by NAS (NAS, 1983, Vol. 5, Drinking Water and Health, National Academy of Sciences Press, Washington, DC.)

Reference -- 50 FR 46936 (11/13/85)

EPA Contact -- Health and Ecological Criteria Division / OST /  
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

#### IV.B.2. MAXIMUM CONTAMINANT LEVEL (MCL) for Drinking Water

Value (status) -- 0.05 mg/L (Interim, 1980)

Considers technological or economic feasibility? -- YES

Discussion -- As an interim measure the U.S. EPA is using the value previously derived by the Public Health Service.

Monitoring requirements -- Ground water systems every three years; surface water systems annually.

Analytical methodology -- Atomic absorption/furnace technique (EPA 206.2; SM 304); atomic absorption/gaseous hydride (EPA 206.3; SM 303E; ASTM D-2972-78B)

Best available technology -- No data available.

Reference -- 45 FR 57332 (08/27/80); 50 FR 46936 (11/13/85)

EPA Contact -- Drinking Water Standards Division / OGWDW /  
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

(CWA) Clean Water Act:

IV.C. CLEAN WATER ACT (CWA)

IV.C.1. AMBIENT WATER QUALITY CRITERIA, Human Health

Water and Fish Consumption --  $2.2E-3$  ug/L

Fish Consumption Only --  $1.75E-2$  ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero.

However, zero may not be attainable at this time, so the recommended criteria represents a  $E-6$  estimated incremental increase of cancer risk over a lifetime.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS  
(202)260-1315 / FTS 260-1315

#### IV.C.2. AMBIENT WATER QUALITY CRITERIA, Aquatic Organisms

##### Freshwater:

Acute -- 3.6E+2 ug/L (Arsenic III)  
Chronic -- 1.9E+2 ug/L (Arsenic III)

##### Marine:

Acute -- 6.9E+1 ug/L (Arsenic III)  
Chronic -- 3.6E+1 ug/L (Arsenic III)

Considers technological or economic feasibility? -- NO

Discussion -- The criteria given are for Arsenic III. Much less data are available on the effects of Arsenic V to aquatic organisms, but the toxicity seems to be less. A complete discussion may be found in the referenced notice.

Reference -- 50 FR 30784 (07/29/85)

EPA Contact -- Criteria and Standards Division / OWRS  
(202)260-1315 / FTS 260-1315

(FIFRA) Federal Insecticide, Fungicide, and Rodenticide Act:  
IV.D. FEDERAL INSECTICIDE, FUNGICIDE, AND RODENTICIDE ACT (FIFRA)  
IV.D.1. PESTICIDE ACTIVE INGREDIENT, Registration Standard

Status -- Issued (1988)

Reference -- Arsenic, Chromium and Chromated Arsenical Compounds Pesticide Registration Standard. June, 1988. [NTIS# PB89-102842]

EPA Contact -- Registration Branch / OPP  
(703)557-7760 / FTS 557-7760

#### IV.D.2. PESTICIDE ACTIVE INGREDIENT, Special Review

Action -- Final regulatory decision - PD4 (1988)

Considers technological or economic feasibility? -- NO

Summary of regulatory action -- Cancellation of specified non-wood uses.  
Registrant of lead arsenate voluntarily canceled 09/87. Registrant of calcium

arsenate voluntarily canceled 02/14/89. Use of sodium arsenate as ant bait canceled on 07/26/89. Criterion of concern: oncogenicity, mutagenicity and teratogenicity. Previous actions: 1) Voluntary cancellation of sodium

arsenite (1978). Voluntary cancellation of two products. Criterion of concern: oncogenicity, mutagenicity and teratogenicity; 2) PD4 (1984). Requires label changes for wood use including a restricted use classification.

Criterion of concern: oncogenicity, mutagenicity and teratogenicity; 3) Voluntary cancellation of copper arsenate (1977). Criterion of concern: oncogenicity.

Reference -- 53 FR 24787 (06/30/88); 43 FR 48267 (10/18/78); 42 FR 18422 (04/07/77); 49 FR 28666 (07/13/84) [NTIS# PB84-241538]; 49 FR 43772 (10/31/84); 50 FR 4269 (01/30/85)

EPA Contact -- Special Review Branch / OPP  
(703)557-7400 / FTS 557-7400

(RCRA) Resource Conservation and Recovery Act:  
IV.F. RESOURCE CONSERVATION AND RECOVERY ACT (RCRA)  
IV.F.1. RCRA APPENDIX IX, for Ground Water Monitoring

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)260-3000 / FTS 260-3000

(CERCLA) Superfund Act:  
IV.G. SUPERFUND (CERCLA)  
IV.G.1. REPORTABLE QUANTITY (RQ) for Release into the Environment

Value (status) -- 1 pound (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The 1-pound RQ for arsenic is based on its potential carcinogenicity. Available data indicate a hazard ranking of high based on a

potency factor of 142.31/mg/kg/day and a weight-of-evidence group A, which corresponds to an RQ of 1 pound. Evidence found in "Water-Related Environmental Fate of 129 Priority Pollutants" (EPA 440/4-79-029a) also indicates that this material, or a constituent of this material, is bioaccumulated to toxic levels in the tissue of aquatic and marine organisms,

and has the potential to concentrate in the food chain. Reporting of releases of massive forms of this hazardous substance is not required if the diameter of the pieces released exceeds 100 micrometers (0.004 inches).

Reference -- 54 FR 33418 (08/14/89)

File 2; Entry 1; Accession No. 1010

(CAS) CAS Registry Number: 7440-39-3

(MAT) Material Name: Barium

(SYN) Synonyms:

Barium;  
UN 1399;  
UN 1400;  
UN 1854

(UPD) Update Date: 08-01-90

(EFF) Effective Date: 10-01-91

(STAT) Status:

STATUS OF DATA FOR Barium

File On-Line 01-31-87

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	08-01-90
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	no data	
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	on-line	06-01-90
Supplementary Data (V.)	no data	

(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Increased blood pressure	NOAEL: 10 mg/L (0.21 mg/kg/day)	3	1	7E-2 mg/kg/day

Subchronic to Chronic Human Drinking Water Studies LOAEL: None

Wones et al., 1990;  
Brenniman and Levy, 1984  
.....

\*Conversion Factors: 10 mg/L x 1.5 L/day/70 kg = 0.21 mg/kg/day

#### I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Wones, R.G., B.L. Stadler and L.A. Frohman. 1990. Lack of effect of drinking water barium on cardiovascular risk factor. Environ. Health Perspect. 85: 1-13.

Brenniman, G.R. and P.S. Levy. 1984. High barium levels in public drinking water and its association with elevated blood pressure. In: Advances in Modern Toxicology IX, E.J. Calabrese, Ed. Princeton Scientific Publications, Princeton NJ. p. 231-249.

No single study considered alone is appropriate to calculate a lifetime RfD for barium. The RfD must be based rather on a weight of evidence approach which takes into account recent findings of the Wones et al. (1990) and Brenniman and Levy (1984) epidemiologic studies as well as the various rodent studies that have been conducted (Perry et al., 1983; McCauley et al., 1985; Schroeder and Mitchener, 1975a,b; Tardiff et al., 1980). Because of the number of studies involved, the complete reference citations are given in the Section VI.

Wones et al. (1990) administered barium (as barium chloride) in the drinking water of 11 healthy male volunteers. Subjects ranged in age from 27 to 61 years and had no previous history of diabetes, hypertension, or cardiovascular disease. Diets were strictly controlled throughout the 10-week study. Subjects were given 1.5 L/day of distilled and charcoal-filtered water containing 0 mg/L barium for weeks 0 to 2; 5 mg/L for weeks 3 to 6, and 10 mg/L for weeks 7 to 10. Blood and urine samples, as well as morning and evening blood pressures, were taken. Electrocardiograms and 24-hour continuous electrocardiographic monitoring were also performed.

There were no changes in systolic or diastolic blood pressures, or serum chemistry, especially total cholesterol, HDL, LDL, triglycerides, potassium or glucose levels. There was an increase in serum calcium levels that was attributed to a decrease in serum albumin levels. This increase, although statistically significant, was considered borderline and not clinically significant. There were also no changes in cardiac cycle as noted by electrocardiograms and no significant arrhythmias. A NOAEL of 10 mg/L was identified in this study which corresponds to 0.21 mg/kg/day, based on an actual consumption rate of 1.5 L/day and a 70-kg body weight.

Brenniman and Levy (1984) conducted a retrospective epidemiology study which compared human mortality and morbidity rates in populations ingesting elevated barium levels (2 to 10 mg/L) in their drinking water to populations ingesting very little or no barium (less than or equal to 0.2 mg/L). Mortality rates for cardiovascular diseases were determined for the years 1971-1975 and were age-adjusted. For the morbidity study, 1175 adult males and 1203 adult females were selected from communities in which the average drinking water

concentration was 7.3 mg/L. Differences in mortality rates from all cardiovascular diseases were significantly higher ( $p < 0.05$ ) in the communities with elevated barium. However, these differences were largely in the 65 and over age group and did not account for confounding variables such as population mobility, or use of water softeners or medication.

Differences in blood pressure, prevalence of hypertension, stroke, and heart and renal disease were also measured between the individuals in the two communities. Data were analyzed using signed ranked test for age-specific rates, the weighted Z test for prevalence rates, and analysis of variance for blood pressures. No significant differences were found in mean systolic and diastolic pressures between the two communities. No significant differences were found when the total populations were broken down by duration (10 years or more), medication, or use of water softeners. Also, the prevalence rates for hypertension, stroke, and heart and kidney disease were not significantly different between the communities.

A concentration of 7.3 mg/L corresponds to a dose of 0.20 mg/kg/day (assuming a 70-kg adult drinks 2 L/day).

#### I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF - 3. According to U.S. EPA guidelines, an uncertainty factor of 10 is applied when a NOAEL from a subchronic human study is employed. However, data are available from chronic human studies which support this NOAEL, as well as several oral chronic animal studies. Therefore, this UF is not considered necessary. In addition, another factor of 10 is used with a human study to protect sensitive individuals. However, the data base supports the finding that the critical effect is hypertension which results from long exposure durations, and that the population most at risk is the adult male. Furthermore, the chosen study is a careful observation of this critical effect in adult males. Because of both the critical study's unique focus and the supporting studies, a 3-fold UF, instead of a 10-fold UF, was chosen as most appropriate to protect for sensitive individuals within that population.

MF - 1.

#### I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

Occupational studies of workers exposed to barium dust have shown that workers develop "baritosis." Affected workers showed no symptoms, no abnormal physical signs, no loss of vital capacity or interference with function, although they had a significantly higher incidence of hypertension.

McCauley et al. (1985) studied the histologic and cardiovascular effects of drinking water containing 0, 10, 100, or 250 mg/L barium for 36 weeks; 0, 1, 10, 100, or 1000 mg/L barium for 16 weeks, or 0, 10, 100, or 250 mg/L (0, 1.4, 14, 35, or 140 mg/kg Ba) barium for 68 weeks on male Sprague-Dawley rats (6/group). Females were exposed to 0 or 250 mg/L for 46 weeks. No significant histologic, carcinogenic, or cardiovascular (including hypertension) effects were observed. No changes were reported in body weight, or food and water consumption in any of the treated animals. Animals treated at the highest dose (1000 mg/L) did exhibit ultrastructural changes in the kidney glomeruli and the presence of myelin figures. No other effects were



reported at any dose level for males or females.

Perry et al. (1983) exposed weanling rats to barium at 1, 10, or 100 ppm in drinking water for up to 16 months (average daily barium doses of 0.051, 0.51, and 5.1 mg/kg, respectively). There were no signs of toxicity at any barium dose level. Systolic blood pressure measurements revealed no increase in animals exposed to 1 ppm for 16 months, an increase of 4 mm Hg ( $p < 0.01$ ) in animals exposed to 10 ppm barium for 16 months, and an increase of 16 mm Hg ( $p < 0.001$ ) in animals exposed to 100 ppm barium for 16 months. The animals in this study were maintained in a special contaminant-free environment and fed a diet designed to reduce exposure to trace metals. It is possible that the restricted intake of certain beneficial metals (e.g., calcium and potassium) may have predisposed the test animals to the hypertensive effects of barium (U.S. EPA, 1985).

Schroeder and Mitchener (1975a,b) exposed rats and mice to 5 mg/L barium in drinking water for a lifetime (approximately 0.25 mg/kg/day for rats and 0.825 mg/kg/day for mice). No adverse effects were observed; however, blood pressure was not measured.

Tardiff et al. (1980) exposed rats to barium at 0, 10, 50, or 250 ppm in drinking water for 4, 8, and 13 weeks. The barium concentrations were approximately 0, 2.75, 13.7, and 66.25 mg/kg/day at the beginning of the study and 0, 1.7, 6.6, and 31.5 mg/kg/day at the end of the study. Although the barium body burden increased with increasing barium dosage, no conclusive signs of barium toxicity were observed in these animals. Blood pressure was not measured.

#### I.A.5. CONFIDENCE IN THE ORAL RfD

Study: Medium  
Data Base: Medium  
RfD: Medium

As previously stated, EPA does not believe that any single study, considered alone, is adequate to calculate an RfD for barium. However, EPA believes that medium confidence can be placed in the total data base used to determine the RfD.

#### I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- U.S. EPA. 1985. Draft Drinking Water Health Effects Criteria Document on Barium. Office of Drinking Water, Washington, DC. NTIS PB 86-118031/AS.

Agency RfD Work Group Review: 07/08/85, 07/22/85, 12/15/87, 05/17/90, 06/21/90

Verification Date: 06/21/90

#### I.A.7. EPA CONTACTS (ORAL RfD)

Kenneth L. Bailey / ODW -- (202)260-5535 / FTS 260-5535  
Linda R. Papa / ODW -- (513)569-7587 / FTS 684-7587

(CAR) Carcinogenicity Assessment:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

This substance/agent has not been evaluated by the U.S. EPA for evidence of human carcinogenic potential.

(REGS) Regulations:

III. HEALTH HAZARD ASSESSMENTS FOR VARIED EXPOSURE DURATIONS

IV. U.S. EPA REGULATORY ACTIONS

IV.A. CLEAN AIR ACT (CAA)

No data available

IV.B. SAFE DRINKING WATER ACT (SDWA)

IV.B.1. MAXIMUM CONTAMINANT LEVEL GOAL (MCLG) for Drinking Water

Value (status) -- 1.5 mg/L (Proposed, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- An MCLG of 1.5 mg/L for barium is proposed based on a provisional DWEL of 1.8 mg/L. A DWEL was calculated from a LOAEL of 5.1 mg/kg/day barium for hypertensinogenic and cardiotoxic effects in rats (16-month drinking water study). An uncertainty factor of 100 (based on minimized exposure to calcium) was applied and consumption of 2 L of water/day was assumed. Data indicate that 83% is the relative source contribution from drinking water. Data were factored in on humans (0.7 mg/day in the diet and 0 mg/day by air).

Reference -- 50 FR 46936 Part IV (11/13/85)

EPA Contact -- Criteria and Standards Division, ODW /  
(202)382-7571 / FTS 382-7571; or Drinking Water Hotline / (800)426-4791

IV.B.2. MAXIMUM CONTAMINANT LEVEL (MCL) for Drinking Water

Value (status) -- 1.0 mg/L (Interim, 1980)

Considers technological or economic feasibility? -- YES

Discussion --

Reference -- 45 FR 57332

EPA Contact -- Kenneth Bailey / Criteria and Standards Division, ODW /  
(202)382-7571 / FTS 382-7571; or Drinking Water Hotline / (800)426-4791

IV.F. RESOURCE CONSERVATION AND RECOVERY ACT (RCRA)

IV.F.1. RCRA APPENDIX IX, for Ground Water Monitoring

Status -- Listed

**Reference -- 52 FR 25942 (07/09/87)**

**EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000**

Option? CAS/71432

File: 11 Count: 1

Option? TYPE 11/2

File 11; Entry 1; Accession No. 1276

(CAS) CAS Registry Number: 71-43-2

(MAT) Material Name: Benzene

(SYN) Synonyms:

Benzene;  
benzol;  
coal naphtha;  
cyclohexatriene;  
phene;  
phenyl hydride;  
polystream;  
pyrobenzol

(UPD) Update Date: 01-01-91

(EFF) Effective Date: 07-01-91

(STAT) Status:

STATUS OF DATA FOR Benzene

File On-Line 03-01-88

Category (section)	Status	Last Revised
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Oral RfD Assessment (I.A.)	pending	
Inhalation RfC Assessment (I.B.)	pending	
Carcinogenicity Assessment (II.)	on-line	01-01-91
Drinking Water Health Advisories (III.A.)	on-line	08-01-90
U.S. EPA Regulatory Actions (IV.)	on-line	08-01-90
Supplementary Data (V.)	no data	

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(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

A risk assessment for this substance/agent will be reviewed by an EPA work group.

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

A risk assessment for this substance/agent is under review by an EPA work group.

(CAR) Carcinogenicity Assessment:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- A; human carcinogen

Basis -- Several studies of increased incidence of nonlymphocytic leukemia from occupational exposure, increased incidence of neoplasia in rats and mice exposed by inhalation and gavage, and some supporting data form the basis for this classification.

II.A.2. HUMAN CARCINOGENICITY DATA

Aksoy et al. (1974) reported effects of benzene exposure among 28,500 Turkish workers employed in the shoe industry. Mean duration of employment was 9.7 years (1-15 year range) and mean age was 34.2 years. Peak exposure was reported to be 210-650 ppm. Twenty-six cases of leukemia and a total of 34 leukemias or preleukemias were observed, corresponding to an incidence of 13/100,000 (by comparison to 6/100,000 for the general population). A follow-up paper (Aksoy, 1980) reported eight additional cases of leukemia as well as evidence suggestive of increases in other malignancies.

In a retrospective cohort mortality study Infante et al. (1977a,b) examined leukemogenic effects of benzene exposure in 748 white males exposed while employed in the manufacturing of rubber products. Exposure occurred

from 1940-1949, and vital statistics were obtained through 1975. A statistically significant increase ( $p$  less than or equal to 0.002) of leukemias was found by comparison to the general U.S. population. There was no evidence of solvent exposure other than benzene. Air concentrations were generally found to be below the recommended limits in effect during the study period.

In a subsequent retrospective cohort mortality study Rinsky et al. (1981) observed seven deaths from leukemia among 748 workers exposed to benzene and followed for at least 24 years (17,020 person-years). This increased incidence was statistically significant; standard mortality ratio (SMR) was 560. For the five leukemia deaths that occurred among workers with more than 5 years exposure, the SMR was 2100. Exposures (which ranged from 10-100 ppm 8-hour TWA) were described as less than the recommended standards for the time period of 1941-1969.

In an updated version of the Rinsky et al. (1981) study, the authors followed the same cohort to 12/31/81 (Rinsky et al., 1987). In his earlier study, cumulative exposure was derived from historic air-sampling data or interpolated estimates based on existing data. Standardized mortality rates ranged from 109 at cumulative benzene exposures under 40 ppm-years and increased monotonically to 6637 (6 cases) at 400 ppm-years or more. The authors found significantly elevated risks of leukemia at cumulative exposures less than the equivalent current standard for occupational exposure which is 10 ppm over a 40-year working lifetime.

Ott et al. (1978) observed three deaths from leukemia among 594 workers followed for at least 23 years in a retrospective cohort mortality study, but the increase was not statistically significant. Exposures ranged from <2 to >25 ppm 8-hour TWA.

Wong et al. (1983) reported on the mortality of male chemical workers who had been exposed to benzene for at least 6 months during the years 1946-1975. The study population of 4062 persons was drawn from seven chemical plants, and jobs were categorized as to peak exposure. Those with at least 3 days/week exposure (3036 subjects) were further categorized on the basis of an 8-hour

Risk estimates based on animal gavage studies are about 5 times higher than those derived from human data. Pharmacokinetic data which could impact the risk assessment are currently being evaluated.

## II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

### II.C.1. SUMMARY OF RISK ESTIMATES

Inhalation Unit Risk --  $8.3E-6$  per (ug/cu.m)

Extrapolation Method -- One-hit (pooled data)

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
-----	-----
E-4 (1 in 10,000)	$1E+1$ ug/cu.m
E-5 (1 in 100,000)	$1E+0$ ug/cu.m
E-6 (1 in 1,000,000)	$1E-1$ ug/cu.m

### II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION EXPOSURE

Species/Strain Tumor Type	Reference
-----	-----
Human/leukemia	Route: Occupational, inhalation Rinsky et al., 1981; Ott et al., 1978; Wong et al., 1983

### II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE)

The unit risk estimate is the geometric mean of four ML point estimates using pooled data from the Rinsky et al. (1981) and Ott et al. (1978) studies, which was then adjusted for the results of the Wong et al. (1983) study. The Rinsky data used were from an updated tape which reports one more case of leukemia than was published in 1981. Equal weight was given to cumulative dose and weighted cumulative dose exposure categories as well as to relative and absolute risk model forms. The results of the Wong et al. (1983) study were incorporated by assuming that the ratio of the Rinsky-Ott-Wong studies to the Rinsky-Ott studies for the relative risk cumulative dose model was the

The slope factor was derived from human data for inhalation exposure as described in section II.C.2. The human respiratory rate was assumed to be 20 cu.m/day, inhalation absorption was taken as 100% and an air concentration of benzene of 1 ppm was taken to equal 3.25 ng/cu.m. The water unit risk was calculated on the assumption that an adult human consumes 2 L water/day.

#### II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

The unit risk estimate is the geometric mean of four ML point estimates using pooled data from the Rinsky et al. (1981) and Ott et al. (1978) studies, which was then adjusted for the results of the Wong et al. (1983) study as described in the additional comments section for inhalation data.

The unit risk should not be used if the water concentration exceeds  $1E+4$  ug/L, since above this concentration the unit risk may not be appropriate.

#### II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

The pooled cohorts were sufficiently large and were followed for an adequate time period. The increases in leukemias were statistically significant and dose-related in one of the studies. Wong et al. (1983) disagrees that exposures reported in Rinsky et al. (1981) were within the recommended standards. For the five leukemia deaths in persons with 5 or more years exposure, the author notes that mean exposure levels (range 15-70 ppm)

exceeded the recommended standard (25 ppm) in 75% of the work locations sampled. A total of 21 unit risk estimates were prepared using 6 models and

various combinations of the epidemiologic data. These range over slightly more than one order of magnitude. A geometric mean of these estimates is  $2.7E-2$ . Regression models give an estimate similar to the geometric mean.

The risk estimate above based on reconsideration of the Rinsky et al. (1981) and Ott et al. (1978) studies is very similar to that of  $2.4E-2$ /ppm

(cited in U.S. EPA, 1980) based on Infante et al. (1977a,b), Ott et al. (1978) and Aksoy et al. (1974). It was felt by the authors of U.S. EPA (1985) that

the exposure assessment provided by Aksoy was too imprecise to warrant inclusion in the current risk estimate.



tively. Likewise male Sprague-Dawley rats exposed by inhalation to 300 ppm benzene were not observed to have increased incidence of neoplasia (Snyder et al., 1981).

Maltoni et al. (1983) treated male and female Sprague-Dawley rats in the following manner. Starting at 13 weeks of age rats were exposed to 200 ppm benzene 4 hours/day, 5 days/week for 7 weeks; 200 ppm 7 hours/day, 5 days/week for 12 weeks; 300 ppm 7 hours/day, 5 days/week for 85 weeks. An 8-hour/day TWA for 5 days/week was calculated to be 241 ppm. A statistically significant increase was noted in hepatomas and carcinomas of the Zymbal gland.

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Numerous investigators have found significant increases in chromosomal aberrations of bone marrow cells and peripheral lymphocytes from workers with exposure to benzene (IARC, 1982). Benzene also induced chromosomal aberrations in bone marrow cells from rabbits (Kissling and Speck, 1973), mice (Meyne and Legator, 1980) and rats (Anderson and Richardson, 1979). Several investigators have reported positive results for benzene in mouse micronucleus assays (Meyne and Legator, 1980). Benzene was not mutagenic in several bacterial and yeast systems, in the sex-linked recessive lethal mutation assay with *Drosophila melanogaster* or in mouse lymphoma cell forward mutation assay.

#### II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

##### II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor --  $2.9E-2$  per (mg/kg)/day

Drinking Water Unit Risk --  $8.3E-7$  per (ug/L)

Extrapolation Method -- One-hit (pooled data)

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
-----	-----
E-4 (1 in 10,000)	$1E+2$ ug/L
E-5 (1 in 100,000)	$1E+1$ ug/L
E-6 (1 in 1,000,000)	$1E+0$ ug/L

##### II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

See table in Section II.C.2.

TWA. The control subjects held jobs at the same plants for at least 6 months but were never subject to benzene exposure. Dose-dependent increases were seen in leukemia and lymphatic and hematopoietic cancer. The incidence of leukemia was responsible for the majority of the increase. It was noted that the significance of the increase is due largely to a less than expected incidence of neoplasia in the unexposed subjects.

Numerous other epidemiologic and case studies have reported an increased incidence or a causal relationship between leukemia and exposure to benzene (IARC, 1982).

#### II.A.3. ANIMAL CARCINOGENICITY DATA

Both gavage and inhalation exposure of rodents to benzene have resulted in development of neoplasia. Maltoni and Scarnato (1979) and Maltoni et al. (1983) administered benzene by gavage at dose levels of 0, 50, 250, and 500 mg/kg bw to 30-40 Sprague-Dawley rats/sex for life. Dose-related increased incidences of mammary tumors were seen in females and of Zymbal gland carcinomas, oral cavity carcinomas and leukemias/lymphomas in both sexes.

In an NTP (1986) study, benzene was administered by gavage doses of 0, 50, 100, or 200 mg/kg bw to 50 F344/N rats/sex or 0, 25, 50, or 100 mg/kg bw to 50 B6C3F1 mice/sex. Treatment was 5 times/week for 103 weeks. Significantly increased incidences ( $p < 0.05$ ) of various neoplastic growths were seen in both sexes of both species. Both male and female rats and mice had increased incidence of carcinomas of the Zymbal gland. Male and female rats had oral cavity tumors, and males showed increased incidences of skin tumors. Mice of both sexes had increased incidence of lymphomas and lung tumors. Males were observed to have harderian and preputial gland tumors and females had tumors of mammary gland and ovary. In general, the increased incidence was dose-related.

Slightly increased incidences of hematopoietic neoplasms were reported for male C57Bl mice exposed by inhalation to 300 ppm benzene 6 hours/day, 5 days/week for 488 days. There was no increase in tumor incidence in male AKR or CD-1 mice similarly exposed to 100 ppm or 100 or 300 ppm benzene, respec-

same as for other model-exposure category combinations and multiplying this ratio by the Rinsky-Ott geometric mean. The age-specific U.S. death rates for 1978 (the most current year available) were used for background leukemia and total death rates. It should be noted that a recently published paper (Rinsky et al., 1987) reported yet another case of leukemia from the study population.

The unit risk should not be used if the air concentration exceeds 100 ug/cu.m, since above this concentration the unit risk may not be appropriate.

#### II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOSURE)

The pooled cohorts were sufficiently large and were followed for an adequate time period. The increases in leukemias were statistically significant and dose-related in one of the studies. Wong et al. (1983) disagrees that exposures reported in Rinsky et al. (1981) were within the recommended standards. For the five leukemia deaths in persons with 5 or more years exposure, the author notes that mean exposure levels (range 15-70 ppm) exceeded the recommended standard (25 ppm) in 75% of the work locations sampled. The risk estimate above based on reconsideration of the Rinsky et al. (1981) and Ott et al. (1978) studies is very similar to that of  $2.4E-2/ppm$  (cited in U.S. EPA, 1980) based on Infante et al. (1977a,b), Ott et al. (1978) and Aksoy et al. (1974). It was felt by the authors of U.S. EPA (1985) that the exposure assessment provided by Aksoy was too imprecise to warrant inclusion in the current risk estimate. A total of 21 unit risk estimates were prepared using 6 models and various combinations of the epidemiologic data. These range over slightly more than one order of magnitude. A geometric mean of these estimates is  $2.7E-2/ppm$ . Regression models give an estimate similar to the geometric mean.

#### II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

##### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1980. Ambient Water Quality Criteria Document for Benzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office (Cincinnati, OH) and Carcinogen Assessment Group (Washington, DC), and the Environmental Research Labs (Corvallis, OR;

Duluth, MN; Gulf Breeze, FL) for the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-018.

U.S. EPA. 1985. Interim Quantitative Cancer Unit Risk Estimates Due to Inhalation of Benzene. Prepared by the Office of Health and Environmental Assessment, Carcinogen Assessment Group, Washington, DC for the Office of Air Quality Planning and Standards, Washington, DC.

U.S. EPA. 1987. Memorandum from J. Orme, HEB, CSD/ODW to C. Vogt, Criteria and Standards Division, ODW, June, 1987.

#### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The 1985 Interim Evaluation was reviewed by the Carcinogen Assessment Group.

The 1987 memorandum is an internal document.

Agency Work Group Review: 03/05/87, 10/09/87

Verification Date: 10/09/87

#### II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

D.L. Bayliss / ORD -- (202)382-5726 / FTS 382-5726

R. McGaughy / ORD -- (202)382-5898 / FTS 382-5898

#### (HA) Hazard Assessment:

##### III. HEALTH HAZARD ASSESSMENTS FOR VARIED EXPOSURE DURATIONS

##### III.A. DRINKING WATER HEALTH ADVISORIES

##### III.A.1. ONE-DAY HEALTH ADVISORY FOR A CHILD

Appropriate data for calculating a One-day HA are not available. It is recommended that the Ten-day HA of 0.235 mg/L used as the One-day HA.

##### III.A.2. TEN-DAY HEALTH ADVISORY FOR A CHILD

Ten-day HA -- 2.35E-1 mg/L

NOAEL -- 2.35 mg/kg/day

UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)

Assumptions -- 1 L/day water consumption for a 10-kg child

Principal Study -- Deichman et al., 1963

Rats were exposed to benzene for 6 hours/day, 4 days/week by inhalation and their hematology was monitored weekly. By the second week of treatment, hematological impairment was observed at the 2659 mg/cu.m exposure concentration and there was some indication, especially in females, that white blood cells were depressed at the 103 mg/cu.m exposure concentration. No effect was seen when animals were exposed to 96 mg/cu.m for up to 4 months. Based on the conditions of exposure and an assumed absorption factor of 50%, a NOAEL of 2.35 mg/kg/day can be calculated.

#### III.A.3. LONGER-TERM HEALTH ADVISORY FOR A CHILD

A Longer-term HA has not been calculated for benzene because of its potent carcinogenicity.

#### III.A.4. LONGER-TERM HEALTH ADVISORY FOR AN ADULT

A Longer-term HA has not been calculated for benzene because of its potent carcinogenicity.

#### III.A.5. DRINKING WATER EQUIVALENT LEVEL / LIFETIME HEALTH ADVISORY

DWEL -- None

Lifetime HA -- None

Benzene is classified in Group A: Human carcinogen. Neither a DWEL nor a Lifetime HA have been calculated for benzene. Refer to Section II of this file for information on the carcinogenicity of this substance.

#### III.A.6. ORGANOLEPTIC PROPERTIES

Odor perception threshold (air) -- 4.9 mg/cu.m.

Odor perception threshold (water) -- 2.0 mg/L.

#### III.A.7. ANALYTICAL METHODS FOR DETECTION IN DRINKING WATER

Analysis of benzene is by a purge-and-trap gas chromatographic procedure used for the determination of volatile aromatic and unsaturated organic compounds in water.

#### III.A.8. WATER TREATMENT

Treatment technologies which will remove benzene from water include granular activated carbon adsorption and air stripping.

### III.A.9. DOCUMENTATION AND REVIEW OF HAS

Deichman, W.B., W.E. MacDonald and E. Bernal. 1963. The hemopoietic toxicity of benzene vapors. Toxicol. Appl. Pharmacol. 5: 201-224.

U.S. EPA. 1985. Drinking Water Criteria Document for Benzene. Office of Drinking Water, Washington, DC. (Final draft)

EPA review of HAS in 1985.

Public review of HAS following notification of availability in October, 1985.

Scientific Advisory Panel review of HAS in January, 1986.

Preparation date of this IRIS summary -- 06/19/87

### III.A.10. EPA CONTACTS

Jennifer Orme / ODW -- (202)382-7586 / FTS 382-7586

Edward V. Ohanian / ODW -- (202)382-7571 / FTS 382-7571

(REGS) Regulations:

#### IV. U.S. EPA REGULATORY ACTIONS

##### IV.A. CLEAN AIR ACT (CAA)

##### IV.A.1. NATIONAL EMISSIONS STANDARDS FOR HAZARDOUS AIR POLLUTANTS (NESHAP)

Considers technological or economic feasibility? -- YES

Discussion -- Benzene has been listed as a hazardous air pollutant under Section 112 of the Clean Air Act. EPA promulgated NESHAP for benzene from equipment leaks on June 6, 1984 (49 FR 23498) and proposed regulations for coke oven by-product plants.

Reference -- 40 CFR Part 61, Subpart J

EPA Contact -- Emissions Standards Division, OAQPS  
(917)541-5571 / FTS 629-5571

##### IV.B. SAFE DRINKING WATER ACT (SDWA)

##### IV.B.1. MAXIMUM CONTAMINANT LEVEL GOAL (MCLG) for Drinking Water

Value (status) -- 0 mg/L (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- An MCLG of zero mg/L for benzene is proposed based on carcinogenic effects. In humans, exposure to benzene is associated with myelocytic anemia, thrombocytopenia and leukemia (acute myelogenous and monocytic leukemia). In animals, an increase in tumors and leukemia have been reported. EPA has classified benzene in Group A: sufficient evidence from epidemiological studies.

Reference -- 50 FR 46880 Part III (11/13/85)

EPA Contact -- Criteria and Standards Division, ODW /  
(202)382-7571 / FTS 382-7571; or Drinking Water Hotline / (800)426-4791

#### IV.B.2. MAXIMUM CONTAMINANT LEVEL (MCL) for Drinking Water

Value (status) -- 5 ug/L (Final, 1987)

Considers technological or economic feasibility? -- YES

Discussion -- The MCL is based on technology and cost factors.

Reference -- 52 FR 25690 (07/08/87)

EPA Contact -- Criteria and Standards Division, ODW /  
(202)382-7571 / FTS 382-7571; or Drinking Water Hotline / (800)426-4791

#### IV.C. CLEAN WATER ACT (CWA)

##### IV.C.1. AMBIENT WATER QUALITY CRITERIA, Human Health

Water and Fish Consumption --  $6.6E-1$  ug/L

Fish Consumption Only --  $4.0E+1$  ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero.

However, zero may not be attainable at this time, so the recommended criteria represents a  $E-6$  estimated incremental increase of cancer risk over a lifetime.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division, OWRS  
(202)475-7315 / FTS 475-7315

##### IV.C.2. AMBIENT WATER QUALITY CRITERIA, Aquatic Organisms

Freshwater:

Acute LEC -- 5.3E+3 ug/L  
Chronic LEC -- None

Marine:

Acute LEC -- 5.1E+3 ug/L  
Chronic LEC -- 7.0E+2 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LECs are given when the minimum data required to derive water quality criteria are not available.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division, OWRS  
(202)475-7315 / FTS 475-7315

IV.F. RESOURCE CONSERVATION AND RECOVERY ACT (RCRA)

IV.F.1. RCRA APPENDIX IX, for Ground Water Monitoring

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

IV.G. SUPERFUND (CERCLA)

IV.G.1. REPORTABLE QUANTITY (RQ) for Release into the Environment

Value (status) -- 10 pounds (Proposed, 1987)

Considers technological or economic feasibility? -- NO

Discussion -- The proposed RQ for benzene is 10 pounds, based on its potential carcinogenicity. The available data indicate a hazard ranking of medium based on a potency factor of 0.27/mg/kg/day and a weight-of-evidence group A, which corresponds to an RQ of 10 pounds.

Reference -- 52 FR 8140 (03/16/87)

EPA Contact -- RCRA/Superfund Hotline



(800)424-9346 / (202)382-3000 / FTS 382-3000

Option? TYPE 12/2

File 12; Entry 1; Accession No. 1454

(CAS) CAS Registry Number: 56-55-3

(MAT) Material Name: Benz[a]anthracene

(SYN) Synonyms:

Benz(a)anthracene;  
benz(a)anthracene;  
Benzanthracene;  
Benzanthrene;  
BENZO(a)ANTHRACENE;  
BENZO(b)PHENANTHRENE;  
Benzoanthracene;  
HSDB 4003;  
NSC 30970;  
RCRA WASTE NUMBER U018;  
Tetraphene;  
1,2-BENZ(a)ANTHRACENE;  
1,2-Benzanthracene;  
1,2-BENZANTHRAZEN [German];  
1,2-BENZANTHRENE;  
1,2-BENZOANTHRACENE;  
2,3-Benzophenanthrene

(UPD) Update Date: 12-01-90

(EFF) Effective Date: 07-01-91

(STAT) Status:

STATUS OF DATA FOR Benz[a]anthracene

File On-Line 12-01-90

Category (section)	Status	Last
Revised		
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Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	
12-01-90		
Drinking Water Health Advisories (III.A.)	no data	

U.S. EPA Regulatory Actions (IV.)

no data

Supplementary Data (V.)

no data

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(CAR) Carcinogenicity Assessment:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen

Basis -- Based on no human data and sufficient data from animal bioassays.

Benz[a]anthracene produced tumors in mice exposed by gavage; intraperitoneal, subcutaneous or intramuscular injection; and topical application.

Benz[a]anthracene produced mutations in bacteria and in mammalian cells, and transformed mammalian cells in culture.

II.A.2. HUMAN CARCINOGENICITY DATA

None. Although there are no human data that specifically link exposure to

benz[a]anthracene to human cancers, benz[a]anthracene is a component of mixtures that have been associated with human cancer. These include coal tar,

soots, coke oven emissions and cigarette smoke (U.S. EPA, 1984, 1990; IARC, 1984; Lee et al., 1976; Brockhaus and Tomingas, 1976).

II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. Benz[a]anthracene administration caused an increase in the

incidence of tumors by gavage (Klein, 1963); dermal application (IARC, 1973);

and both subcutaneous injection (Steiner and Faulk, 1951; Steiner and

Edgecomb, 1952) and intraperitoneal injection (Wislocki et al.,

1986) assays.

A group of male B6AF1/J mice was exposed to gavage solutions containing 3%

benz[a]anthracene in Methocel-Aerosol O.T. (dioctyl ester of sodium sulfo-

succinic acid), 3 doses/week for 5 weeks (total dose of approximately 225

mg/mouse, 500 mg/kg/day) or the vehicle (Klein, 1963). Mice were evaluated

for tumors on days 437-444 and 547 after treatment was initiated. A

statistical analysis was not reported. Increased incidences of pulmonary

adenoma and hepatoma in treated vs. control mice were reported by the authors

at both observation times. The incidence of pulmonary adenoma at 437-444 days

was 37/39 (95%) in treated animals vs. 10/38 (26%) in controls; whereas at 547

days, 19/20 (95%) treated animals and 7/20 (35%) controls had pulmonary

adenomas. The incidence of hepatomas at 437 to 440 days was 18/39 (46%) in

treated animals compared with 0/38 among the vehicle controls. After 547

days, the hepatoma incidences increased to 20/20 for the treated animals

versus 2/20 (10%) for vehicle controls.

Mice (strain and sex not specified) were exposed to a single gavage dose

of 0.5 mg benz[a]anthracene in mineral oil (approximately 17 mg/kg). No

tumors were reported in 13 mice examined 16 months after exposure. In another

part of the study, multiple gavage treatments, 8 or 16 treatments at 3-7 day

intervals over a 16-month period, resulted in forestomach papillomas in 2/27

treated mice compared with 0/16 in vehicle controls (Bock and King, 1959).

Groups of male and female CD-1 mice (n=90-100) received intraperitoneal

injections of benz[a]anthracene in DMSO on days 1, 8, and 15 of age (total

dose = 638 ug/mouse) (Wislocki et al., 1986). Tumors were evaluated in

animals that died spontaneously after weaning and in all remaining animals at

1 year after exposure. In treated male mice, a statistically significant

increase in the incidence of liver adenomas or carcinomas (31/39 treated vs.

2/28 controls) occurred; 25/39 had carcinomas. Female mice did not develop liver tumors. The incidence of pulmonary adenomas or carcinomas in benz[a]anthracene-treated males (6/39, with a majority of adenomas) was increased but not statistically significantly relative to the vehicle controls

(1/28). In the female mice, however, the incidence of pulmonary adenomas was significantly elevated in the treated group (6/32) when compared with vehicle controls (0/31).

Benz[a]anthracene yielded positive results in tests for complete carcinogenicity and initiating activity in skin painting assays in C3H/He, CAF1 and ICR/Ha mouse strains. These studies are reviewed in IARC (1973).

Subcutaneous injection of benz[a]anthracene in tricapyrylin into C57Bl mice (40-50/group) produced injection site sarcomas 9 months after treatment (Steiner and Falk, 1951; Steiner and Edgecomb, 1952). The sarcoma incidences were: uninjected controls, 0/76; tricapyrylin controls, 3/28 (11%); 0.05 mg, 5/43 (12%); 0.2 mg, 11/43 (26%); 1.0 mg, 15/31 (48%); 5.0 mg, 49/145 (34%); and 10 mg, 5/16 (31%). The results of similar experiments in this series were combined (Steiner and Edgecomb, 1952). A statistical analysis of the results was not reported. Survival was roughly equivalent in all groups (70%).

Klein (1952) showed that an intramuscular injection of benz[a]anthracene in combination with 1 or 3% croton oil produced injection site fibrosarcomas and hemangioendotheliomas in Strain A-derived albino mice; 3/24 mice injected with benz[a]anthracene and 1% croton oil and 1/26 mice injected with benz[a]anthracene and 3% croton oil developed tumors. None of the 30 mice injected with benz[a]anthracene and 0.1% croton oil and none of the 30 mice injected with benz[a]anthracene and 5% croton oil developed tumors. In the control groups none of the 35 mice injected only with 1% croton

oil and none of the 32 mice injected only with benz[a]anthracene developed tumors. The survival rate for all groups was roughly equivalent (74%).

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

The results of tests for DNA damage in *Escherichia coli* have not been

positive at concentrations of benz[a]anthracene up to 250 ug/mL and 1000

ug/well (Rosenkrantz and Poirier, 1979; DeFlora et al., 1984). Positive

results were obtained in tests for reverse mutation in five different strains

of *Salmonella typhimurium* and for forward mutation in one strain (McCann et

al., 1975; Coombs et al., 1976; Simmon, 1979; Salamone et al., 1979; Bartsch

et al., 1980; DeFlora et al., 1984; Norpoth et al., 1984; Utesch et al., 1987;

Bos et al., 1988; Kaden et al. 1979).

Benz[a]anthracene produced positive results in an assay for mutations in

*Drosophila melongaster* (Fahmy and Fahmy, 1973).

Tests for DNA damage, mutation, chromosomal effects and cell

transformation in a variety of eukaryotic cell preparations have yielded

mostly positive results. Benz[a]anthracene tested positive for DNA damage in

primary rat hepatocytes and HeLa cells (Probst et al., 1981; Martin et al.,

1978). It also tested positive for forward mutation in Chinese hamster cells,

V79 cells, mouse lymphoma L5178Y cells and rat liver epithelial cells (Slaga

et al., 1978; Krahn and Heidelberger, 1977; Amacher et al., 1980; Amacher and

Turner, 1980; Tong et al., 1981). Benz[a]anthracene tested positive for

chromosomal affects in Chinese hamster ovary cells (Pal, 1981).

Tests for

cell transformation (cell morphology) have yielded positive results in Syrian

hamster embryo cells and mouse prostate C3HG23 cells (Pienta et al., 1977;

DiPaolo et al., 1969, 1971; Marquardt and Heidelberger, 1972).

Current theories on mechanisms of metabolic activation of polycyclic

aromatic hydrocarbons are consistent with a carcinogenic potential for benz[a]anthracene. Benz[a]anthracene has a "bay-region" structure (Jerina et al., 1978). It is metabolized by mixed function oxidases to reactive "bay-region" diol epoxides that are mutagenic in bacteria and tumorigenic in mouse skin painting assays (Booth and Sims, 1974; Wood et al., 1977a,b).

## II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1984. Carcinogen Assessment of Coke Oven Emissions. Office of Health and Environmental Assessment, Washington, DC. EPA 600/6-82-003F. NTIS PB 84-170181.

U.S. EPA. 1990. Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons (PAHs). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. Final Draft. ECAO-CIN-D010, September, 1990.

### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The 1990 Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons has received Agency and external review.

Agency Work Group Review: 02/07/90

Verification Date: 02/07/90

### II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Rita S. Schoeny / ORD -- (513)569-7544 / FTS 684-7544

Robert E. McGaughy / ORD -- (202)382-5889 / FTS 382-5889

Option? TYPE 14.^H/2

File 14; Entry 1; Accession No. 1453

(CAS) CAS Registry Number: 205-99-2

(MAT) Material Name: Benzo[b]fluoranthene

(SYN) Synonyms:

Benz(e)acephenanthrylene;  
B(b)F;  
BENZ(e)ACEPHENANTHRYLENE;  
Benzo(b)fluoranthene;  
Benzo(e)fluoranthene;  
HSDB 4035;  
NSC 89265;  
2,3-BENZFLUORANTHENE;  
2,3-BENZOFUORANTHENE;  
2,3-BENZOFUORANTHRENE;  
3,4-BENZ(e)ACEPHENANTHRYLENE;  
3,4-BENZFLUORANTHENE;  
3,4-Benzofluoranthene

(UPD) Update Date: 12-01-90

(EFF) Effective Date: 07-01-91

(STAT) Status:

STATUS OF DATA FOR Benzo[b]fluoranthene

File On-Line 12-01-90

Category (section)	Status	Last
Revised		
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Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	
12-01-90		
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	no data	
Supplementary Data (V.)	no data	



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(CAR) Carcinogenicity Assessment:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen

Basis -- Based on no human data and sufficient data from animal bioassays.

Benzo[b]fluoranthene produced tumors in mice after lung implantation, intraperitoneal (i.p.) or subcutaneous (s.c.) injection, and skin painting.

II.A.2. HUMAN CARCINOGENICITY DATA

None. Although there are no human data that specifically link exposure to

benzo[b]fluoranthene to human cancers, benzo[b]fluoranthene is a component of

mixtures that have been associated with human cancer. These include coal tar,

soots, coke oven emissions and cigarette smoke (U.S. EPA, 1984, 1990; IARC, 1984).

II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. In a lifetime implant study, 3-month-old female Osborne-

Mendel rats (35/group) received a single lung implant of either 0.1 mg (0.4

mg/kg), 0.3 mg (1.2 mg/kg) or 1 mg (4.1 mg/kg)

benzo[b]fluoranthene in 0.05 mL

of a 1:1 (v:v) mixture of beeswax and trioctanoin

(Deutsch-Wenzel et al.,

1983). Controls consisted of an untreated group and a group receiving an

implant of the vehicle. The median survival times were: 118, 104, 110, 113

and 112 weeks, for the untreated, vehicle control, low-, mid- and high-dose

groups, respectively. The incidences of epidermoid carcinomas and pleomorphic sarcomas in the lung and thorax (combined) were: untreated controls, 0/35; vehicle controls, 0/35; low-dose group, 1/35; mid-dose group, 3/35; and high-dose group, 13/35. These incidences showed a statistically significant dose-response relationship.

Groups of 15-17 male and 17-18 female CD-1 mice received i.p. injections of benzo[b]fluoranthene in DMSO on days 1, 8 and 15 after birth (total dose was approximately 126 ug/mouse) and were sacrificed at 52 weeks of age (LaVoie et al., 1987). A statistically significant increase in the incidence of liver adenomas and hepatomas (combined) occurred in treated males (8/15) relative to vehicle controls (1/17), but not in females. Lung adenomas (2/15 males, 3/17 females) were reported in treated animals, whereas none were found in controls.

Injection site sarcomas occurred in 18/24 survivors of a total of 16 male and 14 female XVIInc/2 mice that received three s.c. injections of benzo[b]fluoranthene (total dose = 2.6 mg) over a period of 2 months (Lacassagne et al., 1963).

Benzo[b]fluoranthene has yielded positive results for complete carcinogenic activity and initiating activity in mouse skin-painting assays. In skin-painting assays groups of 20 female Swiss mice were treated 3 times/week with 0.01, 0.1 or 0.5% solutions of benzo[b]fluoranthene in acetone (Wynder and Hoffmann, 1959). The high dose produced papillomas in 100% of the mice and carcinomas in 90% of the mice within 8 months. The middle dose produced papillomas in 65% and carcinomas in 85% within 12 months, while the low dose produced a papilloma in only 1 animal among 10 survivors at 14 months. No concurrent controls were observed. LaVoie et al. (1982) applied solutions of 0, 10, 30 or 100 ug benzo[b]fluoranthene in 0.1 mL acetone (10

doses, one every other day) to the skins of groups of 20 Crl:CD-1 mice. This regimen was followed by treatment with 2.5 ug 12-O-tetradecanoyl-phorbol-13-acetone (TPA) (a tumor promoter), 3 times/week for 20 weeks. Increases in the percentage of tumor-bearing animals (0, 45, 60, 80) as well as the number of skin tumors/animal (0, 0.9, 2.3, 7.1) appeared to be dose-related. Similar studies by Amin et al. (1985a,b) resulted in comparable elevations of tumor incidence.

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Positive results have been reported for a reverse mutation assay in *Salmonella* TA98 and the results for *Salmonella* TA100 have been positive and not positive (Mossanda et al., 1979; LaVoie et al., 1979; Hermann, 1981; Amin et al., 1985a,b).

Current theories on mechanisms of metabolic activation of polycyclic aromatic hydrocarbons are consistent with a carcinogenic potential for

benzo[b]fluoranthene. Benzo[b]fluoranthene does not have a "classic bay-region" structure (Jerina et al., 1978). It is metabolized by mixed function oxidases to dihydrodiols (Amin et al., 1982). The 9,10-dihydrodiol is tumorigenic in mouse skin-painting assays, suggesting the possible formation of a reactive diol-epoxide (LaVoie et al., 1982).

#### II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

##### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1984. Carcinogen Assessment of Coke Oven Emissions. Office of Health and Environmental Assessment, Washington, DC. EPA 600/6-82-003F. NTIS PB 84-170181.

U.S. EPA. 1990. Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons (PAHs). Prepared by the Office of Health and

Environmental

Assessment, Environmental Criteria and Assessment Office,  
Cincinnati, OH for  
the Office of Drinking Water, Washington, DC. Final Draft.  
ECAO-CIN-D010,  
September, 1990.

II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The 1990 Drinking Water Criteria Document for Polycyclic  
Aromatic

Hydrocarbons has received Agency and external review.

Agency Work Group Review: 02/07/90

Verification Date: 02/07/90

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

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File 3; Entry 1; Accession No. 1461

(CAS) CAS Registry Number: 191-24-2

(MAT) Material Name: Benzo[g,h,i]perylene

(SYN) Synonyms:

Benzo(ghi)perylene;

benzo(ghi)perylene;

HSDB 6177;

NSC 89275;

1,12-Benzoperylene;

1,12-benzperylene

(UPD) Update Date: 12-01-90

(EFF) Effective Date: 10-01-91

(STAT) Status:

STATUS OF DATA FOR Benzo[g,h,i]perylene

File On-Line 12-01-90

Category (section)	Status	Last Revised
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Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	12-01-90
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	no data	
Supplementary Data (V.)	no data	

(CAR) Carcinogenicity Assessment:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity

Basis -- Based on no human data and inadequate animal data from lung implant, skin-painting and subcutaneous injection bioassays.

II.A.2. HUMAN CARCINOGENICITY DATA

None.

### II.A.3. ANIMAL CARCINOGENICITY DATA

Inadequate. Benzo[g,h,i]perylene appeared to increase lung epidermoid tumors when administered with trioctanonin in a lung implant study (Deutsch-Wenzel et al., 1983). Benzo[g,h,i]perylene was tested for complete carcinogenic activity and initiating activity in mouse skin painting assays and did not produce positive results in either type of assay (Wynder and Hoffmann, 1959; Hoffmann and Wynder, 1966; Muller, 1968; Van Duuren et al., 1973). Benzo[g,h,i]perylene did not induce tumor formation when injected subcutaneously (Muller, 1968) and was tested as a cocarcinogen with benzo(a)pyrene (Van Duuren et al., 1973; Van Duuren and Goldschmidt, 1976).

In a lifetime implant study, 3-month-old female Osborne-Mendel rats (34 to 35/group) received a lung implant of benzo[g,h,i]perylene in 0.05 mL of a 1:1 (v:v) mixture of beeswax and trioctanonin (Deutsch-Wenzel et al., 1983). Rats received either 0.16 mg (0.65 mg/kg), 0.83 mg (3.4 mg/kg) or 4.15 mg (17 mg/kg). Controls consisted of an untreated group and a group receiving an implant of the vehicle. Median survival times (weeks) were: untreated controls, 118; vehicle implant controls, 104; 0.16 mg dose, 109; 0.83 mg dose, 114; and 4.15 mg dose, 106. Epidermoid carcinomas in the lung and thorax were observed at the following incidences: 0/35, 0/35, 0/35, 1/35 (3%), and 4/34 (12%) for the untreated controls, vehicle controls, low-, mid-, and high-dose groups, respectively. The apparent increased incidence of tumors was not statistically significant and no distant tumors were seen.

Benzo[g,h,i]perylene was tested as both a complete carcinogen and as a tumor initiator in female Ha/ICR/mil Swiss albino mice (Hoffmann and Wynder, 1966; Wynder and Hoffman, 1959). Two groups of 20 mice received dermal applications of 0.1 or 0.05% benzo[g,h,i]perylene 3 times/week for 1 year. The mice were observed for 3 additional months and then sacrificed. No tumors were observed in the low-dose group and a papilloma was observed in the high-dose group in the tenth month. In a second part of the study, benzo[g,h,i]perylene was applied as an initiator to 30 mice. Ten separate applications of 0.1% each were given over a 2-day period; beginning 28 days later 2.5% croton oil was applied 3 times/week for the remainder of the year. These mice were observed for 3 additional months; 2/27 surviving mice had developed papillomas in this group. A control group of 30 mice received applications of 2.5% croton oil (3 times/week) without an initiator; no tumor or survival data were reported.

The ability of benzo[g,h,i]perylene to act as a cocarcinogen in female ICR/HA mice when combined with benzo(a)pyrene was examined in a series of experiments (Van Duuren et al., 1973; Van Duuren and Goldschmidt, 1976). The mice (50/group) were treated by dermal application with 21 ug benzo[g,h,i]perylene and 5 ug benzo(a)pyrene (in combination) 3 times/week for 1 year. At the end of the experiment, 20/37 mice had developed papillomas and 17/37 had developed squamous cell carcinomas. In the control group, which consisted of three benzo(a)pyrene treatments (5 ug/week), 13/42 mice developed papillomas and 10/42 developed carcinomas (Van Duuren et al., 1973). In the second experiment two doses of benzo[g,h,i]perylene (7 and 21 ug) were applied along with 5 ug benzo(a)pyrene to groups of 50 mice 3 times/week for 368 days.

In the low-dose group 19 mice developed papillomas and 10 carcinomas; in the

high-dose group, 20 mice developed papillomas and 18 carcinomas. No papillomas or carcinomas developed when 21 ug benzo[g,h,i]perylene was applied alone. The individual animal data were not given for this experiment.

In a series of experiments, Muller (1968) investigated the carcinogenicity of benzo[g,h,i]perylene. In the first experiment groups of 50 NMRI mice (sex unspecified) received dermal applications (2 or 3 times/week) of one of a variety of concentrations of benzo[g,h,i]perylene in dichloromethane. A control group receiving only 0.2 mL dichloromethane was also utilized. The study was terminated 675 days after the first application. Survival was approximately the same in all four groups (33%). No skin papillomas or carcinomas developed; however, both benign (0/18 low-, 2/14 mid- and 3/17 high-dose groups) and malignant (3/18 low-, 4/14 mid- and 1/17 high-dose groups) tumors in survivors did occur at other sites (types and sites not specified). In the control group, 3/17 mice developed benign tumors and 4/17 developed malignant tumors at other sites. Dichloromethane is classified B2, a probable human carcinogen.

In a second dermal application study, groups of 50 mice initially were untreated or treated with a single application of either 1 or 2 mg benzo[g,h,i]perylene. In each group repeated dermal applications of 0.2 mL of 0.5% croton oil (2 times/week) followed for 25 weeks. One mouse in the promoter control group and another in the high-dose group developed skin papillomas; 2/28 (0/28), 4/12 (1/12), and 2/21 (1/21) mice developed benign (malignant) tumors at other sites (unspecified) in the control, low- and high-dose groups, respectively. In the third part of the experiment three groups of 50 female NMRI mice received subcutaneous injections of 0 (control), 0.83 or 16.7 mg benzo[g,h,i]perylene suspended in 0.15 mL 10% aqueous gelatin once every 2 weeks for 6 months [total doses, 0, 10 and 200 mg/animal] and observed to sacrifice on day 675 after the first injection. At that time the survival rate was 36% in each group. No tumor was found at the site of injection in any of the animals. For the control, low- and high-dose groups, respectively, 4/50, 5/50, and 4/50 mice had tumors at other sites. In the final part of the experiment four groups of 20 NMRI mice (sex unspecified) were given subcutaneous injections of 0.15 mL 10% aqueous gelatin containing 0 (control), 0.1, 1, or 10 mg suspended benzo[g,h,i]perylene (total doses, 0, 10, or 100 mg/animal) once every 2 weeks for 20 weeks. The animals were observed until spontaneous death. Survival was not adversely affected by treatment with benzo[g,h,i]perylene (the last animal died 22 months after the start of the study). There is no information to indicate if enough animals survived long enough for tumors to be seen. No skin or subcutaneous tumors were found in mice treated with benzo[g,h,i]perylene or gelatin. Few tumors were found in other organs and the incidences in the benzo[g,h,i]perylene-treated groups were not different from those in the gelatin controls. Earlier skin-painting studies are summarized in IARC (1983).

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Benzo[g,h,i]perylene produced positive results in tests for reverse mutation in three strains of *Salmonella typhimurium* and for forward mutation in one strain (Andrews et al., 1978; Mossanda et al., 1979; Salamone et al., 1979; Sakai et al., 1985; Kaden et al., 1979). A test for DNA damage in Chinese hamster ovary cells also yielded positive results (Garrett and Lewtas, 1983).

**II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE**

**None.**

**II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE**

**None.**

**II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)**

**II.D.1. EPA DOCUMENTATION**

**U.S. EPA. 1990. Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons (PAHs). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. Final Draft. ECAO-CIN-D010, September, 1990.**

**II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)**

**The 1990 Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons has received Agency and external review.**

**Agency Work Group Review: 02/07/90**

**Verification Date: 02/07/90**

**II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)**

**Rita S. Schoeny / ORD -- (513)569-7544 / FTS 684-7544**

**Robert E. McGaughy / ORD -- (202)260-5889 / FTS 260-5889**



File 2; Entry 1; Accession No. 1012

(CAS) CAS Registry Number: 7440-41-7

(MAT) Material Name: Beryllium

(SYN) Synonyms:

Beryllium;

Beryllium-9;

Glucinum;

RCRA waste number P015;

UN 1567

(UPD) Update Date: 01-01-91

(EFF) Effective Date: 10-01-91

(STAT) Status:

STATUS OF DATA FOR Beryllium

File On-Line 01-31-87

Category (section)	Status	Last Revised
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Oral RfD Assessment (I.A.)	on-line	09-01-90
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	01-01-91
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	on-line	09-01-90
Supplementary Data (V.)	no data	

(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
-----	-----	-----	---	-----
No adverse effects	NOAEL: 5 ppm in	100	1	5E-3
Rat, Chronic Oral	drinking water (0.54			mg/kg/day
Bioassay	mg/kg bw/day)			
Schroeder and	LOAEL: none			
Mitchner, 1975				
-----				

\*Conversion Factors: 5 ppm (5 mg/L) x 0.035 L/day / 0.325 kg bw = 0.54 mg/kg bw/day

#### I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Schroeder, H.A. and M. Mitchner. 1975. Life-term studies in rats: Effects of aluminum, barium, beryllium and tungsten. J. Nutr. 105: 421-427.

Fifty-two weanling Long-Evans rats of each sex received 0 or 5 ppm beryllium (as BeSO<sub>4</sub>, beryllium sulfate) in drinking water. Exposure was for the lifetime of the animals. At natural death the rats were dissected and gross and microscopic changes were noted in heart, kidney, liver, and spleen. There were no effects of treatment on these organs or on lifespan, urinalysis, serum glucose, cholesterol, and uric acid, or on numbers of tumors. Male rats experienced decreased growth rates from 2 to 6 months of age.

Similar studies were carried out on Swiss (CD strain) mice in groups of 54/sex at doses of approximately 0.95 mg/kg/day (Schroeder and Mitchner, 1975). Female animals showed decreased body weight compared with untreated mice at 6 of 8 intervals. Male mice exhibited slight increases in body weight. These effects were not considered adverse, therefore, 0.95 mg/kg/day is considered a NOAEL.

An unpublished investigation by Cox et al. (1975) indicates a much higher dose level (approximately 25 mg/kg/day) in the diet may be a NOEL.

#### I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF = 100. The uncertainty factor of 100 reflects a factor of 10 each for interspecies conversion and for the protection of sensitive human subpopulations.

MF = 1.

#### I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

This RfD is limited to soluble beryllium salts. Data on the teratogenicity or reproductive effects of beryllium are limited. It has been reported to produce embryoletality and terata in chick embryos (Puzanova et al., 1978).

#### I.A.5. CONFIDENCE IN THE ORAL RfD

Study: Low  
Data Base: Low  
RfD: Low

Confidence in the study is rated as low because only one dose level was administered. Although numerous inhalation investigations and a supporting chronic oral bioassay in mice exist, along with the work by Cox et al. (1975) which indicates that a higher dose level might be a NOEL, these studies are considered as low to medium quality; thus, the data base is given a low confidence rating. The overall confidence in the RfD is low, reflecting the

need for more toxicity data by the oral route.

I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

U.S. EPA. 1985. Drinking Water Criteria Document for Beryllium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.

The 1985 Drinking Water Criteria Document for Beryllium is currently undergoing Agency review.

Agency RfD Work Group Review: 12/02/85

Verification Date: 12/02/85

I.A.7. EPA CONTACTS (ORAL RfD)

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(CAR) Carcinogenicity Assessment:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen.

Basis -- Beryllium has been shown to induce lung cancer via inhalation in rats and monkeys and to induce osteosarcomas in rabbits via intravenous or intramedullary injection. Human epidemiology studies are considered to be inadequate.

II.A.2. HUMAN CARCINOGENICITY DATA

Inadequate. Reported increases, while apparently associated with exposure, did not take a variety of possible confounding factors into account. Wagoner et al. (1980) observed 47 deaths from cancer among 3055 white males employed in beryllium-processing with a median duration of employment of 7.2 months. Among the 2068 followed for 25 years or more, 20 lung cancer deaths were observed. These increased incidences were statistically significant. When lung cancer mortality data became available for 1968-1975, the number of expected deaths was recalculated and the increased incidence was statistically significant only among workers followed 25 years or more (Bayliss, 1980; MacMahon, 1977, 1978). When the number of expected deaths was adjusted for smoking, the increased incidence was no longer significant (U.S. EPA, 1986).

An earlier study of workers from this same beryllium processing plant, and

several studies of workers from this plant combined with workers from other beryllium plants, have reported a statistically significant increased incidence of lung cancer (Bayliss and Wagoner, 1977; Mancuso, 1970, 1979, 1980). No adjustment was made for smoking in these studies, and all were limited in their ability to detect a possible increased incidence of lung cancer because of methodological constraints and deficiencies.

### II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. Based on the evidence for induction of tumors by a variety of beryllium compounds in male and female monkeys and in several strains of rats of both sexes, via inhalation and intratracheal instillation, and the induction of osteosarcomas in rabbits by intravenous or intramedullary injection in multiple studies.

Slight increases in cancer incidence (not statistically significant in comparison with controls) were reported in Long-Evans rats (52/sex/group) administered 5 ppm beryllium sulfate in the drinking water for a lifetime. The authors reported a slight excess of grossly observed tumors in the 5 ppm group (9/33) over controls (4/26) in the male rats. The power of this test to detect a carcinogenic effect was reduced by high mortality (approximately 60% survived a pneumonia epidemic at 20 months) (Schroeder and Mitchener, 1975a). Schroeder and Mitchener (1975b) administered 5 ppm beryllium sulfate in drinking water to Swiss mice (54/sex/group) over a lifetime. A non-statistically significant increase in incidence of lymphoma leukemias were reported in the females (9/52) relative to controls (3/47).

An increase in reticulum cell sarcomas of the lungs was seen in male, but not female Wistar-derived rats administered beryllium sulfate in the diet at 5 and 50 ppm, but not at 500 ppm (Morgareidge et al., 1977). The incidence in males equaled 10/49, 17/35, 16/40 and 12/39 for the control, low, intermediate and high dose groups, respectively. Since the results were published only as an abstract, and since no response was seen at the highest dose, these results are considered to be only suggestive for the induction of cancer via this route.

Osteogenic sarcomas were induced in rabbits by intravenous injection of beryllium compounds in at least 12 different studies and by intramedullary injection in at least four studies (U.S. EPA, 1987). Bone tumors were induced by beryllium oxide, zinc beryllium silicate, beryllium phosphate, beryllium silicate and beryllium metal. No bone tumors were reported to be induced by intravenous injection of beryllium oxide or zinc beryllium silicate in rats or guinea pigs (Gardner and Heslington, 1946). Positive results, however, were reported in mice injected with zinc beryllium silicate, although the numbers were not listed (Cloudman et al., 1949). The sarcomas were generally reported to be quite malignant and metastasized to other organs.

Lung tumors, primarily adenomas and adenocarcinomas, have been induced via the inhalation route in both male and female Sprague-Dawley rats during exposure periods of up to 72 weeks by beryllium sulfate (Reeves et al., 1967), in both male and female Sherman and Wistar rats by beryllium phosphate, beryllium fluoride and zinc beryllium silicate (Schapers, 1961), in male Charles River CR-CD rats by beryl ore (Wagner et al., 1969) and in both male and female rhesus monkeys by beryllium sulfate (Vorwald, 1968). Positive

results were seen in rats exposed to beryllium sulfate at concentrations as low as 2 ug/cu.m (Vorwald, 1968).

Tumors were also induced by intratracheal instillation of metallic beryllium, beryllium-aluminum alloys and beryllium oxide in both Wistar rats and rhesus monkeys. Adenomas, adenocarcinomas and malignant lymphomas were seen in the lungs, with lymphosarcomas and fibrosarcomas present at extrapulmonary sites (Groth et al., 1980; Ishinishi et al., 1980).

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Beryllium sulfate and beryllium chloride have been shown to be nonmutagenic in bacterial and yeast gene mutation assays (Simmon et al., 1979). In contrast, gene mutation studies in Chinese hamster V79 and CHO cells were positive (Miyaki et al., 1979; Hsie et al., 1979). Chromosomal aberrations and sister chromatid exchange were also induced by beryllium in cultured human lymphocytes and Syrian hamster embryo cells (Larramendy et al., 1981).

#### II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

##### II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor -- 4.3 per(mg/kg)/day

Drinking Water Unit Risk -- 1.2E-4 per(ug/L)

Extrapolation Method -- Linearized multistage procedure, extra risk

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
-----	-----
E-4 (1 in 10,000)	8.3E-1 ug/L
E-5 (1 in 100,000)	8.3E-2 ug/L
E-6 (1 in 1,000,000)	8.3E-3 ug/L

##### II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor Type -- gross tumors, all sites combined

Test Animals -- rat/Long-Evans, male

Route -- oral, drinking water

Reference -- Schroeder and Mitchener, 1975a

-----Dose-----		Tumor
Admin- istered	Human Equivalent (mg/kg/day)	Incidence
-----	-----	-----
ppm	mg/kg/day	
-----	-----	
0	0	0
5	0.54	0.09
		4/26
		9/33

### II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

The solubility and speciation of beryllium in air and water media vary, with ambient air characterized by relatively insoluble beryllium compounds such as beryllium oxide and metallic beryllium, and water characterized by more soluble forms. Carcinogenic potency varies according to the form of beryllium present.

Human equivalent doses were calculated using a human body weight of 70 kg, an animal weight of 0.325 kg and length of exposure, experiment and lifespan of 1126 days for treated and control animals.

The unit risk should not be used if the water concentration exceeds  $8.3E+1$  ug/L, since above this concentration the unit risk may not be appropriate.

### II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

The estimate is derived from a study which did not show a significant increase in tumorigenic response. While this study is limited by use of only one non-zero dose group, the occurrence of high mortality and unspecified type and site of the tumors, it was used as the basis of the quantitative estimate because exposure occurred via the most relevant route. Oral risk estimates derived by extrapolation from studies in other species/strains for the intravenous and inhalation routes (also highly uncertain) are within an order of magnitude.

### II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

#### II.C.1. SUMMARY OF RISK ESTIMATES

Inhalation Unit Risk --  $2.4E-3$  per (ug/cu.m)

Extrapolation Method -- Relative risk

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
-----	-----
E-4 (1 in 10,000)	$4E-2$ ug/cu.m
E-5 (1 in 100,000)	$4E-3$ ug/cu.m
E-6 (1 in 1,000,000)	$4E-4$ ug/cu.m

#### II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION EXPOSURE

Beryllium Concentration in Workplace (ug/cu.m)	Fraction of Lifetime	Effective dose (ug/cu.m)	95 percent Upper-bound Estimate of Relative Risk	Unit Risk /ug/cu.m
-----	-----	-----	-----	-----
100	1.00	21.92	1.98	$1.61E-3$

			2.09	1.79E-3
	0.25	5.48	1.98	6.44E-3
			2.09	7.16E-3
1000	1.00	219.18	1.98	1.61E-4
			2.09	1.79E-4
	0.25	54.79	1.98	6.44E-4
			2.09	7.16E-4

### II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE)

Human data were used for the inhalation exposure quantitation despite limitations in the study. Humans are most likely to be exposed by inhalation to beryllium oxide, rather than other beryllium salts. Animal studies by inhalation of beryllium oxide have utilized intratracheal instillation, rather than general inhalation exposure.

Effective dose was determined by adjusting for duration of daily (8/24 hours) and annual (240/365 days) exposure, and the fraction of the lifetime at risk (i.e., time from onset of employment to termination of follow-up). The risk estimates were based on the data of Wagener et al. (1980) in which the smoking adjusted, expected lung cancer deaths were found to range from 13.91 to 14.67, in comparison to 20 observed. Relative risk estimates of 1.36 and 1.44 were derived and the 95% confidence limits of these estimates, 1.98 and 2.09, respectively, were used to estimate the lifetime cancer risk. Note that all of the above estimates are based on one data set using a range of estimated exposure and exposure times. Because of uncertainties regarding workplace beryllium concentration and exposure duration, unit risks were derived using two estimates each of concentration, fraction of lifetime exposed and relative risk. The recommended value is the arithmetic mean of the 8 derived unit risks.

The unit risk should not be used if the air concentration exceeds 4 ug/cu.m, since above this concentration the unit risk may not be appropriate.

### II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOSURE)

The estimate of risk for inhalation exposure was based upon an epidemiologic study having several confounding variables. The estimates of exposure levels and duration were also somewhat uncertain. While a quantitative assessment based on several animal studies resulted in a similar estimate of risk (which increases the confidence somewhat), the quality of the available studies was poor (that is, they were conducted at single dose levels or lacked control groups).

## II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1986. Health Assessment Document for Beryllium. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA 600/8-84-026F.

U.S. EPA. 1987. Drinking Water Criteria Document for Beryllium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. (External Review Draft)

### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The values in 1984 Health Assessment Document for Beryllium received Agency and external review. The 1984 Drinking Water Criteria Document received Agency review.

Agency Work Group Review: 05/04/88, 02/01/89, 12/07/89

Verification Date: 05/04/88 (inhalation); 02/01/89 (oral)

### II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

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David Bayliss / ORD -- (202)260-5726 / FTS 260-5726

(REGS) Regulations:

III. HEALTH HAZARD ASSESSMENTS FOR VARIED EXPOSURE DURATIONS

IV. U.S. EPA REGULATORY ACTIONS

IV.A. CLEAN AIR ACT (CAA)

IV.A.1. NATIONAL EMISSIONS STANDARDS FOR HAZARDOUS AIR POLLUTANTS (NESHAP)

Considers technological or economic feasibility? -- YES

Discussion -- Beryllium was listed as a hazardous air pollutant under section 112 of the CAA in 1971 on the basis that it can cause the chronic lung disease berylliosis. Emission standards promulgated for extraction, ceramic, and propellant plants, foundries, incinerators, and machine shops are 10 g/24 hr or attainment of an ambient concentration near the source of 0.01 ug/cu.m. 30 day average. This ambient concentration was judged adequate to protect the public health with an ample margin of safety. More complex standards were also promulgated for beryllium rocket motor firing. The NESHAPs are now under review, and will consider new health evidence that beryllium may be a carcinogen.

Reference -- 40 CFR Part 61, Subparts C & D

EPA Contact -- Emissions Standards Division, OAQPS  
(917)541-5571 / FTS 629-5571



IV.C. CLEAN WATER ACT (CWA)

IV.C.1. AMBIENT WATER QUALITY CRITERIA, Human Health

Water and Fish Consumption:  $6.8E-3$  ug/L

Fish Consumption Only:  $1.17E-1$  ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. However, zero may not be attainable at this time, so the recommended criterion represent a  $E-6$  estimated incremental increase of cancer risk over a lifetime.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division, OWRS  
(202)475-7315 / FTS 475-7315

IV.C.2. AMBIENT WATER QUALITY CRITERIA, Aquatic Organisms

Freshwater:

Acute LEL --  $1.3E+2$  ug/L

Chronic LEL --  $5.3E+0$  ug/L

Marine: None

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LECs are given when the minimum data required to derive water quality criteria are not available.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division, OWRS  
(202)475-7315 / FTS 475-7315

IV.F. RESOURCE CONSERVATION AND RECOVERY ACT (RCRA)

IV.F.1. RCRA APPENDIX IX, for Ground Water Monitoring

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

IV.G. SUPERFUND (CERCLA)

IV.G.1. REPORTABLE QUANTITY (RQ) for Release into the Environment

Value (status) -- 10 pounds (Proposed, 1987)

Considers technological or economic feasibility? -- NO

Discussion -- The proposed RQ for beryllium is based on potential carcinogenicity. Available data indicate a hazard ranking of medium based on a potency factor of 79.70/mg/kg/day and a weight-of-evidence group B2, which correspond to an RQ of 10 pounds.

Reference -- 52 FR 8140 (03/16/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

Option? CAS/117817

File: 5 Count: 1

Option? TYPE 5/2

File 5; Entry 1; Accession No. 1014

(CAS) CAS Registry Number: 117-81-7

(MAT) Material Name: Bis(2-ethylhexyl)phthalate (BEHP)

(SYN) Synonyms:

BEHP;  
Bis(2-ethylhexyl)-1,2-benzene-dicarboxylate;  
Bis(2-ethylhexyl)phthalate;  
Bisoflex 81;  
Bisoflex DOP;  
Compound 889;  
DAF 68;  
DEHP;  
Di(2-ethylhexyl)orthophthalate;  
Di(2-ethylhexyl)phthalate;  
Dioctyl phthalate;  
Di-sec-octyl phthalate;  
DOP;  
Ergoplast FDO;  
Ethylhexyl phthalate;  
2-Ethylhexyl phthalate;  
Eviplast 80;  
Eviplast 81;  
Fleximel;  
Flexol DOP;  
Flexol plasticizer DOP;  
Good-Rite GP 264;  
Hatcol DOP;  
Hercoflex 260;  
Kodaflex DOP;  
Mollan O;  
NCI- C52733;  
Nuoplaz DOP;  
Octoil;  
Octyl phthalate;  
Palatinol AH;  
Phthalic acid, Bis(2-ethylhexyl) ester;  
Phthalic acid, dioctyl ester;  
Pittsburgh PX-138;  
Platinol DOP;  
RC Plasticizer DOP;  
RCRA waste number U028;  
Reomol D 79P;  
Reomol DOP;  
Sicol 150;  
Staflex DOP;

Truflex DOP;  
Vestinol AH;  
Vinicizer 80;  
Witcizer 312

(UPD) Update Date: 05-01-91

(EFF) Effective Date: 07-01-91

(STAT) Status:  
STATUS OF DATA FOR BEHP

File On-Line 01-31-87

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	05-01-91
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	05-01-90
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	on-line	03-01-88
Supplementary Data (V.)	no data	

(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Increased relative liver weight	NOAEL: none	1000	1	2E-2 mg/kg/day
Guinea Pig Sub-chronic-to-Chronic Oral Bioassay	LOAEL: 0.04% of diet (19 mg/kg bw/day)			
Carpenter et al., 1953				

\*Conversion Factors: none

#### I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Carpenter, C.P., C.S. Weil and H.F. Smyth. 1953. Chronic oral toxicity of di(2-ethylhexyl)phthalate for rats and guinea pigs. Arch. Indust. Hyg. Occup. Med. 8: 219-226.

The following numbers of guinea pigs were fed diets containing BEHP for a period of 1 year: 24 males and 23 females consumed feed containing 0.13% BEHP; 23 males and 23 females consumed feed containing 0.04% BEHP; and 24 males and 22 females were fed the control diet. These dietary levels corresponded to 64 or 19 mg/kg bw/day based on measured food consumption. No treatment-related effects were observed on mortality, body weight, kidney weight, or gross pathology and histopathology of kidney, liver, lung, spleen, or testes. Statistically significant increases in relative liver weights were observed in both groups of treated females (64 and 19 mg/kg bw/day).

Groups of 32 male and 32 female Sherman rats were maintained for 2 years on diets containing either 0.04, 0.13 or 0.4% BEHP (equivalent to 20, 60, and about 195 mg/kg bw/day based on measured food consumption). An F1 group of 80 animals was fed the 0.04% diet for 1 year. Mortality in the F1 treated and control groups was high; 46.2 and 42.7%, respectively, survived to 1 year. There was, however, no effect of treatment on either parental or F1 group mortality, life expectancy, hematology, or histopathology of organs. Both parental and F1 rats receiving the 0.4% BEHP diet were retarded in growth and had increased kidney and liver weights.

It appears that guinea pigs offer the more sensitive animal model for BEHP toxicity. A LOAEL in this species is determined to be 19 mg/kg/day.

#### I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF = 1000. Factors of 10 each were used for interspecies variation and for protection of sensitive human subpopulations. An additional factor of 10 was used since the guinea pig exposure was longer than subchronic but less than lifetime, and because, while the RfD is set on a LOAEL, the effect observed was considered to be minimally adverse.

MF = 1.

#### I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

Dietary levels of 0, 0.01, 0.1, and 0.3% BEHP (greater than 99% pure) were administered to male and female CD-1 mice that were examined for adverse fertility and reproductive effects using a continuous breeding protocol. BEHP was a reproductive toxicant in both sexes significantly decreasing fertility

and the proportion of pups born alive per litter at the 0.3% level, and inducing damage to the seminiferous tubules. BEHP has been observed to be

both fetotoxic and teratogenic (Singhe, 1972; Shiot and Nishimura, 1982).

#### I.A.5. CONFIDENCE IN THE ORAL RfD

Study: Medium  
Data Base: Medium  
RfD: Medium

The study by Carpenter et al. (1953) utilized sufficient numbers of guinea pigs and measured multiple endpoints. The fact that there were only two con-

centrations of BEHP tested precludes a rating higher than medium. Since there are corroborating chronic animal bioassays, the data base is likewise rated

medium. Medium confidence in the RfD follows.

#### I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

The RfD has been reviewed by the RfD Work Group. Documentation may be found in the meeting notes of 01/22/86.

Agency RfD Work Group Review: 01/22/86

Verification Date: 01/22/86

#### I.A.7. EPA CONTACTS (ORAL RfD)

Michael L. Dourson / ORD -- (513)569-7544 / FTS 684-7544

Christopher T. DeRosa / ORD -- (513)569-7534 / FTS 684-7534

(CAR) Carcinogenicity Assessment:

#### II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

##### II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

##### II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen.

Basis -- Orally administered DEHP produced significant dose-related increases in liver tumor responses in rats and mice of both sexes.

#### II.A.2. HUMAN CARCINOGENICITY DATA

Inadequate. Thiess et al. (1978) conducted a mortality study of 221 DEHP production workers exposed to unknown concentrations of DEHP for 3 months to 24 years. Workers were followed for a minimum of 5 to 10 years (mean follow-up time was 11.5 years). Eight deaths were reported in the exposed population. Deaths attributable to pancreatic carcinoma (1 case) and uremia (1 case in which the workers also had urethral and bladder papillomas) were significantly elevated in workers exposed for >15 years when compared to the corresponding age groups in the general population. The study is limited by a short follow-up period and unqualified worker exposure. Results are considered inadequate for evidence of a causal association.

#### II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. In an NTP (1982) study, 50 male and 50 female Fisher 344 rats per group were fed diets containing 0, 6000 or 12,000 ppm DEHP for 103 weeks. Similarly, groups of 50 male and 50 female B6C3F1 mice were given 0, 3000 or 6000 ppm DEHP in the diet for 103 weeks. Animals were killed and examined histologically when moribund or after 105 weeks. No clinical signs of toxicity were observed in either rats or mice. A statistically significant increase in the incidence of hepatocellular carcinomas and combined incidence of carcinomas and adenoma were observed in female rats and both sexes of mice.

The combined incidence of neoplastic nodules and hepatocellular carcinomas was statistically significantly increased in the high-dose male rats. A positive dose response trend was also noted.

Carpenter et al. (1953) found no malignant tumors in treated groups of 32 male and 32 female Sherman rats. Animals were given 400, 1300 or 4000 ppm DEHP in the diet for 1 year and reduced to a maximum of 8 males and 8 females and treated for another year. Controls, F1 and 4000 ppm groups were sacrificed after being maintained on control or 4000 ppm diets for 1 year. Only 40 to 47% of the animals in each group, including F1 animals, survived 1 year. Thus, an insufficient number of animals were available for a lifetime evaluation.

Carpenter et al. (1953) did not find a carcinogenic effect in guinea pigs and dogs exposed to 1300 or 4000 ppm DEHP. Both guinea pigs and dogs were terminated after 1 year of exposure. The treatment and survival periods for these animals were considerably below their lifetimes.

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Studies indicate that DEHP is not a direct acting mutagen in either a forward mutation assay in *Salmonella typhimurium* (Seed, 1982) or the rec assay in *Bacillus subtilis* (Tomita et al., 1982). DEHP did not induce mutations in a modified reverse mutation plate incorporation assay in *Salmonella* strains TA100 and TA98 at concentrations up to 1000 ug/plate in the presence or absence of S9 hepatic homogenate (Kozumbo et al., 1982). MEHP, the monoester form of DEHP and a metabolite is positive in the rec assay and in the reverse mutation assay in *Salmonella*. In the absence of exogenous metabolism MEHP produced chromosomal aberrations and sister chromatid exchanges in V79 cells. Both DEHP and MEHP induced chromosomal aberrations and morphological transformation in cultured fetal Syrian hamster cells exposed in utero (Tomita et al., 1982). Chromosomal effects were not found in CHO mammalian cells (Phillips et al., 1982) exposed to DEHP. DEHP was weakly positive with metabolic activation in only one of several studies testing for mutagenic activity at the thymidine kinase locus in L5178Y mouse lymphoma cells (Ashby et al., 1985). DEHP is a potent inducer of hepatic peroxisomal enzyme activity (Ganning et al., 1984).

#### II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

##### II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor --  $1.4E-2/\text{mg/kg/day}$

Drinking Water Unit Risk --  $4.0E-7/\text{ug/L}$

Extrapolation Method -- Linearized multistage procedure, extra risk

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
-----	-----
E-4 (1 in 10,000)	$3E+2 \text{ ug/L}$
E-5 (1 in 100,000)	$3E+1 \text{ ug/L}$
E-6 (1 in 1,000,000)	$3 \text{ ug/L}$



## II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor Type -- Mouse/B6C3F1, male  
Test Animals -- hepatocellular carcinoma and adenoma  
Route -- oral, diet  
Reference -- NTP, 1982

Admin- istered (ppm)	Dose ----- Human Equivalent (mg/kg/day)	Tumor Incidence
0	0	14/50
3000	32	25/48
6000	65	29/50

## II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

In this study powdered rodent meal was provided in such a way that measured food consumption could include significant waste and spillage rather than true food intake. For this reason a standard food consumption rate of 13% mouse body weight was used in the dose conversion.

DEHP is hydrolyzed to monoesters including MEHP (Pollack et al., 1985; Lhuguenot et al., 1985; Kluwe, 1982). Although several species of animals have been determined to excrete glucuronide conjugates of monoethylhexyl phthalate (MEHP) upon exposure to DEHP, rats do not (Taraka et al., 1975; Williams and Blanchfield, 1975; Albro et al., 1982).

Slope factors based on combined hepatocellular carcinoma and neoplastic nodule incidences were  $4.5E-3$ /mg/kg/day for female rats,  $3.2E-3$ /mg/kg/day for male rats. A slope factor based on hepatocellular adenomas or carcinomas in female mice is  $1.0E-2$ /mg/kg/day.

The unit risk should not be used if the water concentration exceeds  $4E+4$  ug/L, since above this concentration the slope factor may differ from that stated.

## II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

An adequate number of animals was observed and a statistically significant increase in incidence of liver tumors was seen in both sexes and

were dose dependent in both sexes of mice and female rats. A potential source of variability in the NTP study is the possibility of feed scattering. The above calculations are based on standard food consumption rates for mice (13% of body weight) and rats (5% of body weight).

## II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1988. Drinking Water Criteria Document for Phthalic Acid Esters. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. (External Review Draft).

### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The values in the 1988 Drinking Water Criteria Document for Phthalic Acid Esters (External Review Draft) have received Agency review.

Agency Work Group Review: 08/26/87; 10/07/87

Verification Date: 10/07/87

### II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Robert Vanderslice / ODW -- (202)475-6711 / FTS 475-6711

Annette Gatchett / ORD -- (513)569-7813 / FTS 684-7813

(REGS) Regulations:

III. HEALTH HAZARD ASSESSMENTS FOR VARIED EXPOSURE DURATIONS

IV. U.S. EPA REGULATORY ACTIONS

IV.A. CLEAN AIR ACT (CAA)

No data available

IV.C. CLEAN WATER ACT (CWA)

IV.C.1. AMBIENT WATER QUALITY CRITERIA, Human Health

Water and Fish Consumption:  $1.5E+4$  ug/L

Fish Consumption Only:  $5E+4$  ug/L

Considers technological or economic feasibility? -- NO

Discussion --

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division, OWRS  
(202)475-7315 / FTS 475-7315

#### IV.C.2. AMBIENT WATER QUALITY CRITERIA, Aquatic Organisms

##### Freshwater:

Acute LEC --  $9.4E+2$  ug/L  
Chronic LEC --  $3E+0$  ug/L

##### Marine:

Acute LEC --  $2.944E+3$  ug/L  
Chronic LEC --  $3.4E+0$  ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LECs are given when the minimum data required to derive water quality criteria are not available.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division, OWRS  
(202)475-7315 / FTS 475-7315

#### IV.F. RESOURCE CONSERVATION AND RECOVERY ACT (RCRA)

##### IV.F.1. RCRA APPENDIX IX, for Ground Water Monitoring

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

#### IV.G. SUPERFUND (CERCLA)

##### IV.G.1. REPORTABLE QUANTITY (RQ) for Release into the Environment

Value (status) -- 100 pounds (Proposed, 1987)

Considers technological or economic feasibility? -- NO

Discissuion -- The 100-pound RQ is based on assessment for potential carcinogenicity. Available data indicate a hazard ranking of low based on a potency factor of 0.015/mg/kg/day and weight-of-evidence group B2, which corresponds to an RQ of 100 pounds.

Reference -- 52 FR 8140 (03/16/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

Option? TYPE 5/2

File 5; Entry 1; Accession No. 1141

(CAS) CAS Registry Number: 7440-43-9

(MAT) Material Name: Cadmium

(SYN) Synonyms:

C.I. 77180;

Cadmium;

KADMIUM

(UPD) Update Date: 03-01-91

(EFF) Effective Date: 07-01-91

(STAT) Status:

STATUS OF DATA FOR Cadmium

File On-Line 03-31-87

Category (section)	Status	Last Revised
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Oral RfD Assessment (I.A.)	on-line	10-01-89
Inhalation RfC Assessment (I.B.)	pending	
Carcinogenicity Assessment (II.)	on-line	03-01-91
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	on-line	06-01-90
Supplementary Data (V.)	no data	

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(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
-----	-----	-----	-----	-----

Significant proteinuria	NOAEL (water): 0.005 mg/kg/day	10	1	5E-4 mg/kg/day (water)
Human studies involving chronic exposures	NOAEL (food): 0.01 mg/kg/day	10	1	1E-3 mg/kg/day (food)

U.S. EPA, 1985

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\*Conversion Factors: See text for discussion

#### I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

U.S. EPA. 1985. Drinking Water Criteria Document on Cadmium. Office of Drinking Water, Washington, DC. (Final draft)

A concentration of 200 ug cadmium (Cd)/gm wet human renal cortex is the highest renal level not associated with significant proteinuria (U.S. EPA, 1985). A toxicokinetic model is available to determine the level of chronic human oral exposure (NOAEL) which results in 200 ug Cd/gm wet human renal cortex; the model assumes that 0.01% day of the Cd body burden is eliminated per day (U.S. EPA, 1985). Assuming 2.5% absorption of Cd from food or 5% from water, the toxicokinetic model predicts that the NOAEL for chronic Cd exposure is 0.005 and 0.01 mg Cd/kg/day from water and food, respectively (i.e., levels which would result in 200 ug Cd/gm wet weight human renal cortex). Thus, based on an estimated NOAEL of 0.005 mg Cd/kg/day for Cd in drinking water and an UF of 10, an RfD of 0.0005 mg Cd/kg/day (water) was calculated; an equivalent RfD for Cd in food is 0.001 mg Cd/kg/day (see Section VI.A. for references).

#### I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF = 10. This uncertainty factor is used to account for intrahuman variability to the toxicity of this chemical in the absence of specific data on sensitive individuals.

MF = 1.

#### I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

Cd is unusual in relation to most, if not all, of the substances for which an oral RfD has been determined in that a vast quantity of both human and

animal toxicity data are available. The RfD is based on the highest level of Cd in the human renal cortex (i.e., the critical level) not associated with significant proteinuria (i.e., the critical effect). A toxicokinetic model has been used to determine the highest level of exposure associated with the lack of a critical effect. Since the fraction of ingested Cd that is absorbed appears to vary with the source (e.g., food vs. drinking water), it is necessary to allow for this difference in absorption when using the toxicokinetic model to determine an RfD.

#### I.A.5. CONFIDENCE IN THE ORAL RfD

Study: Not applicable  
Data Base: High  
RfD: High

The choice of NOAEL does not reflect the information from any single study. Rather, it reflects the data obtained from many studies on the toxicity of cadmium in both humans and animals. These data also permit calculation of pharmacokinetic parameters of cadmium absorption, distribution, metabolism and elimination. All of this information considered together gives high confidence in the data base. High confidence in either RfD follows as well.

#### I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

U.S. EPA. 1985. Drinking Water Criteria Document on Cadmium. Office of Drinking Water, Washington, DC. (Final draft)

Agency RfD Work Group Review: 05/15/86, 08/19/86, 09/17/87, 12/15/87, 01/20/88, 05/25/88

Verification Date: 05/25/88

#### I.A.7. EPA CONTACTS (ORAL RfD)

Ken Bailey / ODW -- (202)382-5535 / FTS 382-5535

Warren Banks / OWRS -- (202)382-7893 / FTS 382-7893

#### I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

A risk assessment for this substance/agent is under review by an EPA work group.

(CAR) Carcinogenicity Assessment:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B1; probable human carcinogen

Basis -- Limited evidence from occupational epidemiologic studies of cadmium is consistent across investigators and study populations. There is sufficient evidence of carcinogenicity in rats and mice by inhalation and intramuscular and subcutaneous injection. Seven studies in rats and mice wherein cadmium salts (acetate, sulfate, chloride) were administered orally have shown no evidence of carcinogenic response.

II.A.2. HUMAN CARCINOGENICITY DATA

Limited. A 2-fold excess risk of lung cancer was observed in cadmium smelter workers. The cohort consisted of 602 white males who had been employed in production work a minimum of 6 months during the years 1940-1969. The population was followed to the end of 1978. Urine cadmium data available for 261 workers employed after 1960 suggested a highly exposed population. The authors were able to ascertain that the increased lung cancer risk was probably not due to the presence of arsenic or to smoking (Thun et al., 1985). An evaluation by the Carcinogen Assessment Group of these possible confounding factors has indicated that the assumptions and methods used in accounting for them may not be valid. As the SMRs observed were low and there is a lack of clear cut evidence of a causal relationship of the cadmium exposure only, this study is considered to supply only limited evidence of human carcinogenicity.

An excess lung cancer risk was also observed in three other studies which were, however, compromised by the presence of other carcinogens (arsenic, smoking) in the exposure or by a small population (Varner, 1983; Sorahan and Waterhouse, 1983; Armstrong and Kazantzis, 1983).

Four studies of workers exposed to cadmium dust or fumes provided evidence of a statistically significant positive association with prostate cancer (Kipling and Waterhouse, 1967; Lemen et al., 1976; Holden, 1980; Sorahan and Waterhouse, 1983), but the total number of cases was small in each study. The Thun et al. (1985) study is an update of an earlier study (Lemen et al., 1976)



and does not show excess prostate cancer risk in these workers. Studies of human ingestion of cadmium are inadequate to assess carcinogenicity.

#### II.A.3. ANIMAL CARCINOGENICITY DATA

Exposure of Wistar rats to cadmium as cadmium chloride at concentrations of 12.5, 25 and 50 ug/cu.m for 18 months, with an additional 13-month observation period, resulted in significant increases in lung tumors (Takenaka et al., 1983). Intratracheal instillation of cadmium oxide did not produce lung tumors in Fischer 344 rats but rather mammary tumors in females and tumors at multiple sites in males (Sanders and Mahaffey, 1984). Injection site tumors and distant site tumors (for example, testicular) have been reported by a number of authors as a consequence of intramuscular or subcutaneous administration of cadmium metal and chloride, sulfate, oxide and sulfide compounds of cadmium to rats and mice (U.S. EPA, 1985). Seven studies in rats and mice where cadmium salts (acetate, sulfate, chloride) were administered orally have shown no evidence of a carcinogenic response.

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Results of mutagenicity tests in bacteria and yeast have been inconclusive. Positive responses have been obtained in mutation assays in Chinese hamster cells (Dom and V79 lines) and in mouse lymphoma cells (Casto, 1976; Ochi and Ohsawa, 1983; Oberly et al., 1982).

Conflicting results have been obtained in assays of chromosomal aberrations in human lymphocytes treated in vitro or obtained from exposed workers.

Cadmium treatment in vivo or in vitro appears to interfere with spindle formation and to result in aneuploidy in germ cells of mice and hamsters (Shimada et al., 1976; Watanabe et al., 1979; Gilliavod and Leonard, 1975).

#### II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

Not available. There are no positive studies of orally ingested cadmium suitable for quantitation.

## II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

### II.C.1. SUMMARY OF RISK ESTIMATES

Inhalation Unit Risk --  $1.8E-3$  per (ug/cu.m)

Extrapolation Method -- Two stage; only first affected by exposure; extra risk

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
-----	-----
E-4 (1 in 10,000)	$6E-2$ ug/cu.m
E-5 (1 in 100,000)	$6E-3$ ug/cu.m
E-6 (1 in 1,000,000)	$6E-4$ ug/cu.m

### II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION EXPOSURE

Tumor Type -- lung, trachea, bronchus cancer deaths

Test Animals -- human/white male

Route -- inhalation, exposure in the workplace

Reference -- Thun et al., 1985

		No. of Expected		Observed No.
		Lung, Trachea and		of Deaths
Cumulative	24 hour/	Bronchus Cancers	(lung, trachea,	
Exposure	Median	ug/cu.m	Assuming No	bronchus
(mg/day/cu.m)	Observation	Equivalent	Cadmium Effect	cancers)
-----				
less than or equal to 584	280	168	3.77	2
585-2920	1210	727	4.61	7
greater than or equal to 2921	4200	2522	2.50	7

The 24-hour equivalent = median observation  $\times 10E-3 \times 8/24 \times 1/365 \times 240/365$ .

### II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE)

The unit risk should not be used if the air concentration exceeds 6 ug/cu.m, since above this concentration the unit risk may not be appropriate.

#### II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOSURE)

The data were derived from a relatively large cohort. Effects of arsenic and smoking were accounted for in the quantitative analysis for cadmium effects.

An inhalation unit risk for cadmium based on the Takenaka et al. (1983) analysis is  $9.2E-2$  per (ug/cu.m). While this estimate is higher than that derived from human data [ $1.8E-3$  per (ug/cu.m)] and thus more conservative, it was felt that the use of available human data was more reliable because of species variations in response and the type of exposure (cadmium salt vs. cadmium fume and cadmium oxide).

#### II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

##### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1985. Updated Mutagenicity and Carcinogenicity Assessment of Cadmium: Addendum to the Health Assessment Document for Cadmium (May 1981, EPA 600/B-81-023). EPA 600/B-83-025F.

##### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The Addendum to the Cadmium Health Assessment has received both Agency and external review.

Agency Work Group Review: 11/12/86

Verification Date: 11/12/86

##### II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

William E. Pepelko / ORD -- (202)382-5904 / FTS 382-5904

David Bayliss / ORD -- (202)382-5726 / FTS 382-5726

(REGS) Regulations:

III. HEALTH HAZARD ASSESSMENTS FOR VARIED EXPOSURE DURATIONS

IV. U.S. EPA REGULATORY ACTIONS

IV.A. CLEAN AIR ACT (CAA)

IV.A.1. CAA REGULATORY DECISION

Action -- Intent to list under Section 112

Considers technological or economic feasibility? -- NO

Discussion -- Cadmium is a probable human carcinogen (IARC category 2A) and according to EPA's preliminary risk assessment from ambient air exposures,

public health risks are significant (3-7 cancer cases/year and maximum lifetime individual risks of 0.003. Thus, EPA indicated that it intends to add cadmium to the list of hazardous air pollutants for which it intends to establish emission standards under section 112(b)(1)(A) of the Clean Air Act.

The EPA will decide whether to add cadmium to the list only after studying possible techniques that might be used to control emissions and further assessing the public health risks. The EPA will add cadmium to the list if emission standards are warranted.

Reference -- 50 FR 42000 (10/16/85)

EPA Contact -- Emissions Standards Division, OAQPS  
(919)541-5571 / FTS 629-5571

#### IV.B. SAFE DRINKING WATER ACT (SDWA)

##### IV.B.1. MAXIMUM CONTAMINANT LEVEL GOAL (MCLG) for Drinking Water

Value (status) -- 0.005 mg/L (Proposed, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- An MCLG of 0.005 mg/L for cadmium is proposed based on a provisional DWEL of 0.018 mg/L and drinking water contribution (plus aquatic organism) of 25%. A DWEL of 0.018 mg/L was calculated from a LOAEL of 0.352 mg/day for renal toxicity in humans (calculated), with an uncertainty factor of 10 applied and consumption of 2 L of water/day assumed.

Reference -- 50 FR 46936 Part IV (11/13/85)

EPA Contact -- Criteria and Standards Division, ODW /  
(202)382-7571 / FTS 382-7571; or Drinking Water Hotline / (800)426-4791

##### IV.B.2. MAXIMUM CONTAMINANT LEVEL (MCL) for Drinking Water

Value (status) -- 0.01 mg/L (Interim, 1980)

Considers technological or economic feasibility? -- YES

Discussion --

Reference -- 45 FR 57332

EPA Contact -- Kenneth Bailey / Criteria and Standards Division, ODW /  
(202)382-7571 / FTS 382-7571; or Drinking Water Hotline / (800)426-4791

#### IV.C. CLEAN WATER ACT (CWA)

##### IV.C.1. AMBIENT WATER QUALITY CRITERIA, Human Health

Water and Fish Consumption:  $1E+1$  ug/L

Fish Consumption Only: None

Considers technological or economic feasibility? -- NO

Discussion -- The criteria is the same as the existing standard for drinking water.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division, OWRS  
(202)475-7315 / FTS 475-7315

##### IV.C.2. AMBIENT WATER QUALITY CRITERIA, Aquatic Organisms

#### Freshwater:

Acute --  $3.9E+0$  ug/L (1-hour average)  
Chronic --  $1.1E+0$  ug/L (4-day average)

#### Marine:

Acute --  $4.3E+1$  ug/L (1-hour average)  
Chronic --  $9.3E+0$  ug/L (4-day average)

Considers technological or economic feasibility? -- NO

Discussion -- The freshwater criteria are hardness dependent. Values given here are calculated at a hardness of 100 mg/L  $CaCO_3$ . A complete discussion can be found in the referenced notice.

Reference -- 50 FR 30784 (07/29/85)

EPA Contact -- Criteria and Standards Division, OWRS  
(202)475-7315 / FTS 475-7315

IV.D. FEDERAL INSECTICIDE, FUNGICIDE, AND RODENTICIDE ACT (FIFRA)  
IV.D.1. PESTICIDE ACTIVE INGREDIENT, Registration Standard

None

IV.D.2. PESTICIDE ACTIVE INGREDIENT, Special Review

Action -- Final regulatory action - PD4 (1987)

Considers technological or economic feasibility? -- YES

Summary of regulatory action -- The basis for selection of the final regulatory option is presented in Position Document 4.

Reference -- 52 FR 31076 (08/19/87)

EPA Contact -- Special Review Branch, OPP / (703)557-7400 / FTS 557-7400

IV.F. RESOURCE CONSERVATION AND RECOVERY ACT (RCRA)  
IV.F.1. RCRA APPENDIX IX, for Ground Water Monitoring

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

IV.G. SUPERFUND (CERCLA)  
IV.G.1. REPORTABLE QUANTITY (RQ) for Release into the Environment

Value (status) -- 10 pounds (Proposed, 1987)

Considers technological or economic feasibility? -- NO

Discussion -- The proposed RQ for cadmium is 10 pounds, based on potential carcinogenicity. Available data indicate a hazard ranking of medium, based on a potency factor of 57.87/mg/kg/day and weight-of-evidence group B1, which corresponds to an RQ of 10 pounds. Cadmium has also been found to bioaccumulate in the tissues of aquatic and marine organisms, and has the potential to concentrate in the food chain.

Reference -- 52 FR 8140 (03/16/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

Option? TYPE 6/2

File 6; Entry 1; Accession No. 1020

(CAS) CAS Registry Number: 56-23-5

(MAT) Material Name: Carbon tetrachloride

(SYN) Synonyms:

Acritet;  
Benzinoform;  
Carbona;  
Carbon chloride;  
Carbon tet;  
Carbon tetrachloride;  
Carbo tetrachloride;  
Czterochlorek wegla;  
ENT 4,705;  
Fasciolin;  
Flukoids;  
Freon 10;  
Halon 104;  
Mecatorina;  
Methane tetrachloride;  
Methane, tetrachloro-;  
Necatorina;  
Necatorine;  
Perchloromethane;  
R 10;  
Tetrachloorkoolstof;  
Tetrachloormetaan;  
Tetrachlorkohlenstoff, tetra;  
Tetrachlormethan;  
Tetrachlorocarbon;  
Tetrachloromethane;  
Tetrachlorure de carbone;  
Tetrachorkohlenstoff uvasol;  
Tetraclorometano;  
Tetracloruro di carbonio;  
Tetrafinol;  
Tetraform;  
Tetrasol;  
Univerm;  
Ventox;  
Vermoestricid;  
WLN: GXGGG.

(UPD) Update Date: 06-01-91

(EFF) Effective Date: 07-01-91



(STAT) Status:  
STATUS OF DATA FOR Carbon tetrachlor

File On-Line 01-31-87

Category (section) -----	Status -----	Last Revised -----
Oral RfD Assessment (I.A.)	on-line	06-01-91
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	06-01-91
Drinking Water Health Advisories (III.A.)	on-line	08-01-90
U.S. EPA Regulatory Actions (IV.)	on-line	06-01-91
Supplementary Data (V.)	no data	

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(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.A.1. ORAL RfD SUMMARY

Critical Effect -----	Experimental Doses* -----	UF -----	MF ---	RfD -----
Liver lesions	NOAEL: 1 mg/kg/day (converted to 0.71	1000	1	7E-4 mg/kg/day
Subchronic Rat Gavage Study	mg/kg/day)  LOAEL: 10 mg/kg/day (converted to 7.1			
Bruckner et al., 1986	mg/kg/day)			

---

\*Conversion Factors:  $1 \text{ mg/kg/day (NOAEL)} \times 5/7 = 0.71 \text{ mg/kg/day (5 day/week dosing regimen)}$

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Bruckner, J.V., W.F. MacKenzie, S. Muralidhara, R. Luthra, G.M. Kyle and D. Acosta. 1986. Oral toxicity of carbon tetrachloride: Acute, subacute and

subchronic studies in rats. Fund. Appl. Toxicol. 6(1): 16-34.

Male Sprague-Dawley rats were given 1, 10, or 33 mg carbon tetrachloride/kg/day by corn oil gavage, 5 days/week for 12 weeks. Liver lesions, as evidenced by mild centrilobular vacuolization and statistically significant increases in serum sorbitol dehydrogenase activity, were observed at the 10 and 33 mg/kg/day doses in a dose-related manner. Therefore, the LOAEL was established at 10 mg/kg/day (converted to 7.1 mg/kg/day) and the NOAEL was 1 mg/kg/day (converted to 0.71 mg/kg/day).

#### I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF = 1000. UF allows for interspecies and intrahuman variability and extrapolation from subchronic to chronic duration of exposure.

MF = 1.

#### I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

A 1983 draft of the Bruckner et al. (1986) study was used as the basis for the RfD by the RfD Work Group at a 05/20/85 verification meeting. When this study was subsequently published (Bruckner et al., 1986), no change to the verified value was required.

Subchronic studies in mice gavaged with carbon tetrachloride in corn oil (Condie et al., 1986; Hayes et al., 1985) support the critical effect and the magnitude of the NOAEL and LOAEL found in the rat studies. Additional studies (Alumot et al., 1976; NCI, 1976) in rats lend moderate support to the choice of a NOAEL in the chosen rat study.

#### I.A.5. CONFIDENCE IN THE ORAL RfD

Study: High  
Data Base: Medium  
RfD: Medium

The principal study was well conducted and good dose-response was observed in the liver, which is the target organ for carbon tetrachloride toxicity; thus, high confidence was assigned. Four additional subchronic studies support the RfD, but reproductive and teratology endpoints are not well investigated; thus, the data base rates a medium confidence. Medium confidence in the RfD follows.

#### I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

U.S. EPA. 1985. Drinking Water Criteria Document for Carbon Tetrachloride.  
Office of Drinking Water, Washington, DC.

Public review of RfD following ODW proposal of RMCL in June 1984.

Science Advisory Board review of RfD on January 14, 1986.

Agency Work Group Review: 05/20/85

Verification Date: 05/20/85

I.A.7. EPA CONTACTS (ORAL RfD)

Krishan Khanna / OW -- (202)382-7588 / FTS 382-7588

Michael L. Dourson / ORD -- (513)569-7544 / FTS 684-7544

(CAR) Carcinogenicity Assessment:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen

Basis -- Carcinogenicity in rats, mice, and hamsters

II.A.2. HUMAN CARCINOGENICITY DATA

Inadequate. There have been three case reports of liver tumors developing after carbon tetrachloride exposure. Several studies of workers (Milham, 1976; Blair et al., 1979) who may have used carbon tetrachloride have suggested that these workers may have an excess risk of cancer.

II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. Carbon tetrachloride has produced hepatocellular carcinomas in rats, mice, and hamsters, the species evaluated to date.

Hepatocellular carcinomas developed in Osborne-Mendel, Japanese, and Wistar rats, but not Sprague-Dawley or Black rats, following s.c. injection of carbon tetrachloride. Hyperplastic nodules were noted in Buffalo rats treated s.c. (Reuber and Glover, 1967a,b, 1970). Sensitivity varied among strains, and trends in incidence appeared inversely related to severity of

cirrhosis.

Fifty Osborne-Mendel rats/sex were administered carbon tetrachloride by corn oil gavage at 47 and 94 mg/kg/injection for males and 80 and 159 mg/kg for females 5 times/week for 78 weeks. At 110 weeks, only 7/50 high-dose males and 14/50 high-dose females survived; 14/50 low-dose males and 20/50 low-dose females survived. The incidence of hepatocellular carcinomas was increased in animals exposed to carbon tetrachloride as compared with pooled colony controls. The apparent decrease in the incidence of hepatocellular carcinomas in high-dose female rats compared with the low-dose females (1/14 vs. 4/20, respectively) was attributed by the authors to increased lethality before tumors could be expressed (NCI, 1976a,b, 1977).

In this same study, using the same dosing schedule, male and female B6C3F1 mice received 1250 or 2500 mg/kg carbon tetrachloride. The incidences of hepatocellular carcinomas in males were 5/77, 49/49, and 47/48 in the control, low- and high-dose groups, respectively, and 1/80, 40/40, and 43/45 in the control, low- and high-dose groups, respectively.

Carbon tetrachloride administered by gavage has also been shown to produce neoplastic changes in livers of five additional strains of mice (C3H, A, Y, C, and L) (Andervont, 1958; Edwards, 1941; Eschenbrenner and Miller 1943; Edwards and Dalton, 1942; Edwards et al., 1942). In the last study, 56 male and 19 female L mice, which have a low incidence of spontaneous hepatomas, were treated with 0.1 mL of 40% carbon tetrachloride 2 or 3 times/week over 4 months, for a total of 46 treatments. Animals were killed 3 to 3.5 months after the last treatment. The combined hepatoma incidence of treated male mice was 47% (7/15 vs. 2/71 in the untreated male controls); treated females showed an incidence of 38% (3/8 vs. 0/81 in the untreated female controls).

As part of a larger study of liver carcinogens, Della Porta et al. (1961) treated Syrian golden hamsters (10/sex/dose) with carbon tetrachloride by gavage, weekly for 30 weeks. For the first 7 weeks, 0.25 mL of 0.05% carbon

tetrachloride in corn oil was administered; this dose was halved for the remainder of the exposure period. All animals were observed for an additional 25 weeks. All of the 10 hamsters that were killed or dying between weeks 43 and 55 had liver cell carcinomas, compared with 0 in controls.

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Carbon tetrachloride was not mutagenic to either *S. typhimurium* or *E. coli* (McCann et al., 1975; Simmon et al., 1977; Uehleke et al., 1976). At low concentrations, carbon tetrachloride did not produce chromatid or chromosomal aberrations in an epithelial cell line derived from rat liver (Dean and Hodson-Walker, 1979). In vivo unscheduled DNA synthesis assays have likewise been negative in male Fischer 344 rats (Mirsalis and Butterworth, 1980; Mirsalis et al., 1982). Carbon tetrachloride produced mitotic recombination and gene conversion in *S. cerevisiae*, but only at concentrations which reduced viability to 10% (Callen et al., 1980). Carbon tetrachloride may be metabolized to reactive intermediates capable of binding to cellular nucleophilic macromolecules. Negative responses in bacterial mutagenicity assays may have been due to inadequate metabolic activation in the test systems.

#### II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

##### II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor --  $1.3E-1$  per (mg/kg)/day

Drinking Water Unit Risk --  $3.7E-6$  per (ug/L)

Extrapolation Method -- Linearized multistage procedure, extra risk

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
-----	-----
E-4 (1 in 10,000)	3E+1 ug/L
E-5 (1 in 100,000)	3E+0 ug/L
E-6 (1 in 1,000,000)	3E-1 ug/L

#### II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor Type -- Hepatocellular carcinomas/hepatomas  
Route -- oral, gavage

Species/Strain	---- Dose ----	Tumor	Reference
----------------	----------------	-------	-----------

Tumor Type	Administered	Human Equivalent	Incidence	Unit Risk	
	mg/day	mg/kg/day		per (ug/L)	
Hamster/Syrian,	0	0	0/80	3.4E-5	Della
male and female	0.95	1.02	10/19		Porta et al., 1961
Mouse/L, male	0	0	2/152	9.4E-6	Edwards
and female	15	2.3	34/73		et al., 1942
Mouse/B6C3F1,	0	0	6/157	1.8E-6	NCI,
male and female	21	55.4	89/89		1976a,b,
	42	110.8	90/93		1977
Rat/Osborne-Mendel					NCI,
M, F	0	0	0/37	3.1E-7	1976a,b,
M	11	4.5	2/45		1977
F	18	7.4	4/46		
M	21	8.7	2/47		
F	36	14.9	1/30		

#### II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

A geometric mean was calculated from the unit risks derived from the four data sets above. Della Porta et al. (1961) did not report controls in this study, but did give incidence rate for vehicle controls in an earlier study. Animal doses are TWA.

The unit risk should not be used if the water concentration exceeds  $3E+3$  ug/L, since above this concentration the  $S^0$ Qat low doses, presumably because early mortality at higher doses precluded tumor formation. The studies lacked pharmacokinetic data. However, a common biological mechanism, cell death and regeneration, leading to development of the same tumor type, was suggested by observations in all the studies. Since the risk estimates from these data (across 3-4 species and strains) only vary by 2 orders of magnitude, a geometric mean was derived as the risk estimate to accommodate the several study deficiencies.



## II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

### II.C.1. SUMMARY OF RISK ESTIMATES

Inhalation Unit Risk --  $1.5E-5$  per (ug/cu.m)

Extrapolation Method -- Linearized multistage procedure, extra risk

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
-----	-----
E-4 (1 in 10,000)	$7E+0$ ug/cu.m
E-5 (1 in 100,000)	$7E-1$ ug/cu.m
E-6 (1 in 1,000,000)	$7E-2$ ug/cu.m

### II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION EXPOSURE

The inhalation risk estimates were calculated from the oral exposure data in Section II.B.2.

### II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE)

Inhalation risk was calculated assuming an air intake of 20 cu.m/day and 40% absorption rate by humans (U.S. EPA, 1984). This absorption coefficient was based on 30% inhalation in monkeys, and 30% and 57-65% inhalation in humans. A range of estimates of unit risk for inhalation exposures for the four studies cited above was determined, with  $1.5E-5$  per (ug/cu.m) calculated as the geometric mean for the unit risk.

The unit risk should not be used if the air concentration exceeds  $7E+2$  ug/cu.m, since above this concentration the unit risk may not be appropriate.

### II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOSURE)

See II.B.4.

## II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1984. Health Assessment Document for Carbon Tetrachloride. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. EPA 600/8/82-001F.

#### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The 1984 Health Assessment Document for Carbon Tetrachloride received Agency and external review.

Agency Work Group Review: 11/12/86, 12/04/86

Verification Date: 12/04/86

#### II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Jean C. Parker / ORD -- (202)382-5898 / FTS 382-5898

Arthur Chiu / ORD -- (202)382-5898 / FTS 382-5898

#### (HA) Hazard Assessment:

##### III. HEALTH HAZARD ASSESSMENTS FOR VARIED EXPOSURE DURATIONS

##### III.A. DRINKING WATER HEALTH ADVISORIES

##### III.A.1. ONE-DAY HEALTH ADVISORY FOR A CHILD

One-day HA --  $4E+0$  mg/L

NOAEL -- 40 mg/kg/day

UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)

Assumptions -- 1 L/day water consumption for a 10-kg child

Principal Study -- Bruckner et al., 1986

Rats were administered single oral doses of carbon tetrachloride. Doses of 80 mg/kg and higher caused changes in liver enzymes (BUN, GPT, SDH, OCT) and histopathologic liver and kidney changes. A dose of 40 mg/kg produced no effects and is identified as the NOAEL.

##### III.A.2. TEN-DAY HEALTH ADVISORY FOR A CHILD

Ten-day HA --  $1.6E-1$  mg/L

LOAEL -- 16 mg/kg/day

UF -- 1000 (allows for interspecies and intrahuman variability with the use of a LOAEL from an animal study)

Assumptions -- 1 L/day water consumption for a 10-kg child

Principal Study -- Bruckner et al., 1986

Rats were administered nine doses of carbon tetrachloride by gavage over an 11-day period. The lowest dose tested (20 mg/kg/day) produced significant changes in serum enzyme levels and hepatic midzonal vacuolation. Higher doses caused more extensive liver damage. A LOAEL of 16 mg/kg/day is established after adjustment for the treatment schedule.

### III.A.3. LONGER-TERM HEALTH ADVISORY FOR A CHILD

#### LONGER-TERM HEALTH ADVISORY FOR A CHILD

Longer-term (Child) HA --  $7.1\text{E-}2$  mg/L

NOAEL -- 0.71 mg/kg/day

UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)

Assumptions -- 1 L/day water consumption for a 10-kg child

Principal Study -- Bruckner et al., 1986

Rats were administered carbon tetrachloride by gavage, 5 times weekly for 12 weeks, at doses of 1, 10, or 33 mg/kg/day. Doses of 10 and 33 mg/kg/day were hepatotoxic (changes in serum enzyme levels, centrilobular vacuolation, and necrosis). The NOAEL of 1 mg/kg/day, based on a 7 days/week dosing regimen, is equivalent to 0.71 mg/kg/day.

### III.A.4. LONGER-TERM HEALTH ADVISORY FOR AN ADULT

Longer-term (Adult) HA --  $2.5\text{E-}1$  mg/L

NOAEL -- 0.71 mg/kg/day

UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)

Assumptions -- 2 L/day water consumption for a 70-kg adult

Principal Study -- Bruckner et al., 1986 (study described in III.A.3.)

### III.A.5. DRINKING WATER EQUIVALENT LEVEL / LIFETIME HEALTH ADVISORY

DWEL --  $2.5\text{E-}2$  mg/L

Assumptions -- 2 L/day water consumption for a 70-kg adult

RfD Verification Date -- 07/08/85 (see Section I.A. in this file)

Lifetime HA -- None

Note: Carbon tetrachloride is considered to be a probable human carcinogen.

Refer to Section II of this file for information on the carcinogenicity of this substance.

Principal Study (DWEL) -- Bruckner et al., 1986 (This study was used in the derivation of the oral chronic RfD; see Section I.A.2.)

### III.A.6. ORGANOLEPTIC PROPERTIES

Odor perception threshold -- 0.52 mg/L.

### III.A.7. ANALYTICAL METHODS FOR DETECTION IN DRINKING WATER

Analysis of carbon tetrachloride is by a purge-and-trap gas chromatographic procedure used for the determination of volatile organohalides in drinking water. Confirmatory analysis is by mass spectrometry.

### III.A.8. WATER TREATMENT

Treatment techniques which will remove carbon tetrachloride from drinking water include granular activated carbon adsorption, boiling, and air stripping. Conventional treatment processes (coagulation, sedimentation, filtration), even when augmented by the addition of powdered activated carbon, provide little removal of carbon tetrachloride.

### III.A.9. DOCUMENTATION AND REVIEW OF HAS

Bruckner, J.V., W.F. MacKenzie, S. Muralidhara, R. Luthra, G.M. Kyle and D. Acosta. 1986. Oral toxicity of carbon tetrachloride: Acute, subacute and subchronic studies in rats. Fund. Appl. Toxicol. 6(1): 16-34.

U.S. EPA. 1985. Final Draft of the Drinking Water Criteria Document on Carbon Tetrachloride. Office of Drinking Water, Washington, DC.

EPA review of HAS in 1985.

Public review of HAS following notification of availability in October, 1985.

Scientific Advisory Panel review of HAS in January, 1986.

Preparation date of this IRIS summary -- 06/22/87

### III.A.10. EPA CONTACTS

Jennifer Orme / ODW -- (202)382-7586 / FTS 382-7586

Edward V. Ohanian / ODW -- (202)382-7571 / FTS 382-7571

(REGS) Regulations:

IV. U.S. EPA REGULATORY ACTIONS

IV.A. CLEAN AIR ACT (CAA)

IV.A.1. CAA REGULATORY DECISION

Action -- Intent to list under Section 112

Considers technological or economic feasibility? -- NO

Discussion -- EPA's preliminary risk assessment from ambient air exposures indicates that public health risks are significant (about 70 cases/year in the U.S.). Because carbon tetrachloride is extremely stable in the atmosphere, these risks are due to a worldwide buildup of carbon tetrachloride caused by emissions from the U.S. as well as other countries. Since these risks were considered significant, EPA indicated that it intends to add carbon tetrachloride to the list of hazardous air pollutants for which it intends to establish emission standards under section 112(b)(1)(A) of the Clean Air Act.

The EPA will decide whether to add carbon tetrachloride to the list only after studying possible techniques that might be used to control emissions, and further assessing the public health risks. The EPA will add carbon tetrachloride to the list if emission standards are warranted. This decision did not consider the role of carbon tetrachloride in reducing stratospheric ozone. This issue is being evaluated separately and will consider the effect of a number of trace gases on stratospheric ozone.

Reference -- 50 FR 32621 (08/13/85)

EPA Contact -- Emissions Standards Division, OAQPS  
(919)541-5571 / FTS 629-5571

IV.B. SAFE DRINKING WATER ACT (SDWA)

IV.B.1. MAXIMUM CONTAMINANT LEVEL GOAL (MCLG) for Drinking Water

Value (status) -- 0 mg/L (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- An MCLG of 0 mg/L for carbon tetrachloride is proposed based on carcinogenic effects. Carbon tetrachloride has been shown to be carcinogenic

in rats, mice, and hamsters through oral exposure. Hepatocellular carcinomas in several studies have been observed. EPA has classified carbon tetrachloride in Group B2: sufficient evidence in animals and inadequate evidence in humans.

Reference -- 50 FR 46880 Part III (11/13/85)

EPA Contact -- Krishan Khanna / Criteria and Standards Division, OW / (202)382-7588 / FTS 382-7588; or Drinking Water Hotline / (800)426-4791

#### IV.B.2. MAXIMUM CONTAMINANT LEVEL (MCL) for Drinking Water

Value (status) -- 5 ppb (Final, 1987)

Considers technological or economic feasibility? -- YES

Discussion --

Reference -- 52 FR 25690 (07/08/87)

EPA Contact -- Krishan Khanna / Criteria and Standards Division, OW / (202)382-7588 / FTS 382-7588; or Drinking Water Hotline / (800)426-4791

#### IV.C. CLEAN WATER ACT (CWA)

##### IV.C.1. AMBIENT WATER QUALITY CRITERIA, Human Health

Water and Fish Consumption:  $4.0E-1$  ug/L

Fish Consumption Only:  $6.94E+0$  ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For maximum protection from the potential carcinogenic properties of this chemical, the ambient concentration should be zero. However, zero may not be attainable at this time so the recommended criteria represents a E-6 estimated incremental increase in cancer risk over a lifetime.

Reference -- 45 FR 791318 (11/28/80)

EPA Contact -- Criteria and Standards Division, OWRS  
(202)475-7315 / FTS 475-7315

##### IV.C.2. AMBIENT WATER QUALITY CRITERIA, Aquatic Organisms

**Freshwater:**

Acute LEC -- 3.52E+4 ug/L  
Chronic -- None

**Marine:**

Acute LEC -- 5.0E+4 ug/L  
Chronic -- None

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LECs are given when the minimum data required to derive water quality criteria are not available.

Reference -- 45 FR 791318 (11/28/80)

EPA Contact -- Criteria and Standards Division, OWRS  
(202)475-7315 / FTS 475-7315

**IV.D. FEDERAL INSECTICIDE, FUNGICIDE, AND RODENTICIDE ACT (FIFRA)**  
**IV.D.1. PESTICIDE ACTIVE INGREDIENT, Registration Standard**

None

**IV.D.2. PESTICIDE ACTIVE INGREDIENT, Special Review**

Action -- Registration voluntarily canceled

Considers technological or economic feasibility? -- Not applicable

Summary of regulatory action -- For specific details on the Special Review process for this active ingredient please call the EPA Contact.

Reference -- None

EPA Contact -- Special Review Branch, OPP / (703)557-7400 / FTS 557-7400

**IV.F. RESOURCE CONSERVATION AND RECOVERY ACT (RCRA)**  
**IV.F.1. RCRA APPENDIX IX, for Ground Water Monitoring**

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

IV.G. SUPERFUND (CERCLA)

IV.G.1. REPORTABLE QUANTITY (RQ) for Release into the Environment

Value (status) --10 pounds (Proposed, 1987)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ for carbon tetrachloride is based on potential carcinogenicity. Available data indicate a hazard ranking of medium based upon a potency factor of 59.9/mg/kg/day and assignment to weight-of-evidence group B2. This corresponds to an RQ of 10 pounds.

Reference -- 52 FR 8140 (03/16//87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000



Captured 6/11/92

1 - IRIS

IRSN - 27

DATE - 920604

UPDT - 06/04/92, 52 fields

STAT - Oral RfD Assessment (RDO) on-line 03/01/88

STAT - Inhalation RfC Assessment (RDI) pending

STAT - Carcinogenicity Assessment (CAR) pending 05/01/92

STAT - Drinking Water Health Advisories (DWA) on-line 11/01/90

STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92

IRH - 03/01/88 RDO Critical effect added

IRH - 03/01/88 HADV Health Advisory added

IRH - 08/01/89 REFS Bibliography on-line

IRH - 08/01/90 RCRA EPA contact changed

IRH - 10/01/90 RDI Inhalation RfC now under review

IRH - 11/01/90 HADV Full Health Advisory summary added

IRH - 01/01/92 RDO Secondary contact changed

IRH - 01/01/92 EXSR Regulatory actions updated

IRH - 05/01/92 CAR Carcinogenicity assessment now under review

RLEN - 15079

NAME - Chromium(III)

RN - 16065-83-1

SY - 7440-47-3

SY - CHROMIC ION

SY - CHROMIUM

SY - Chromium(III)

SY - CHROMIUM (III) ION

SY - CHROMIUM, ION

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RDO -

o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
No effects observed	NOEL: 5% Cr2O3 in diet 5 days/week for	100	10	1E+0
Rat Chronic Feeding Study	600 feedings (1800 g/kg bw average total dose)		mg/kg/day (as an insoluble salt)	
Ivankovic and Preussmann, 1975	LOAEL: none			

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\*Dose Conversion Factors & Assumptions: 1800 g Cr2O3/kg bw x 1000 mg/g x 0.6849 Cr/g Cr2O3 / 600 feeding days x 5 feeding days/7 days = 1468 mg/kg/day

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o ORAL RFD STUDIES :

Ivankovic, S. and R. Preussmann. 1975. Absence of toxic and carcinogenic effects after administration of high doses of chromic oxide pigment in sub-acute and long-term feeding experiments in rats. Food Cosmet. Toxicol. 13: 347-351.

Groups of 60 male and female rats were fed chromic oxide ( $\text{Cr}_2\text{O}_3$ ) baked in bread at dietary levels of 0, 1, 2, or 5%, 5 days/week for 600 feedings (840 total days). The primary purpose of this study was to assess the carcinogenic potential of  $\text{Cr}_2\text{O}_3$ . Body weight and food consumption were monitored. The average total amounts of ingested  $\text{Cr}_2\text{O}_3$  were given as 360, 720, and 1800 g/kg bw for the 1, 2, and 5% treatment groups, respectively. The animals were maintained on control diets following termination of exposure until they became moribund or died. All major organs were examined histologically. Other toxicologic parameters were not mentioned explicitly, but may have included some or all of those described for the accompanying subchronic study (see below). No effects due to  $\text{Cr}_2\text{O}_3$  treatment were observed at any dose level.

Ivankovic and Preussmann (1975) also treated rats (both sexes, 12-19 rats/group) at dietary levels of 0, 2, or 5%  $\text{Cr}_2\text{O}_3$  in bread, 5 days/week for 90 days. Food consumption and body weight were monitored. Toxicologic parameters included serum protein, bilirubin, hematology, urinalysis, organ weights, and histopathology. The only effects observed were reductions (12-37%) in the absolute weights of the livers and spleens of animals in the high-dose group. Organ weights relative to body weight were not reported. The high dose is equivalent to 1400 mg/kg/day (dose converted using reported data).

Other subchronic oral studies show no indication of adverse effects attributable to trivalent chromium compounds, but dose levels were considerably lower.

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#### o ORAL RFD UNCERTAINTY :

UF = 100. The factor of 100 represents two 10-fold decreases in mg/kg bw/day dose that account for both the expected interhuman and interspecies variability to the toxicity of the chemical in lieu of specific data.

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#### o ORAL RFD MODIFYING FACTOR :

MF = 10. The additional modifying factor of 10 is adopted to reflect uncertainty in the NOEL because: 1) the effects observed in the 90-day study were not explicitly addressed in the 2-year study and, thus, the highest NOAEL in the 2-year study may be a LOAEL; 2) the absorption of chromium is low (<1%) and is influenced by a number of factors; thus, a considerable potential variation in absorption exists; and 3) animals were allowed to die naturally after feeding stopped (2 years) and only then was histology performed.

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#### o ORAL RFD COMMENTS :

This RfD is limited to metallic chromium (III) of insoluble salts. Examples of insoluble salts include chromic III oxide ( $\text{Cr}_2\text{O}_3$ ) and chromium III sulfate [ $\text{Cr}_2(\text{SO}_4)_3$ ].

Very limited data suggest that Cr III may have respiratory effects on humans. No data on chronic or subchronic effects of inhaled Cr III in animals can be found. Adequate teratology data do not exist, but reproductive effects are not seen at dietary levels of 5%  $\text{Cr}_2\text{O}_3$ .

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o ORAL RFD CONFIDENCE :

Study: Low  
Data Base: Low  
RfD: Low

The principal study is rated low because of the lack of explicit detail on study protocol and results. Low confidence in the data base reflects the lack of high-dose supporting data. The low confidence in the RfD reflects the foregoing, but also reflects the lack of an observed effect level. Thus, the RfD, as given, should be considered conservative, since the MF addresses only those factors which might lower the RfD.

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o ORAL RFD SOURCE DOCUMENT :

U.S. EPA. 1984. Health Effects Assessment for Trivalent Chromium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, OHEA for the Office of Solid Waste and Emergency Response.

The ADI in the 1984 Health Effects Assessment document received an Agency review with the help of two external scientists.

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o REVIEW DATES : 11/21/85, 02/05/86  
o VERIFICATION DATE : 11/21/85  
o EPA CONTACTS :

Michael L. Dourson / ORD -- (513)569-7544 / FTS 684-7544

Robert Bruce / ORD -- (513)569-7553 / FTS 684-7553

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RDI -

o INHALATION RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

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HAONE-

NOTE: All chromium HAs are based on total chromium (III and VI).

Appropriate data for calculating a One-day HA are not available. It is recommended that the Ten-day HA of 1.4 mg/L be used as the One-day HA.

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HATEN-

NOTE: All chromium HAs are based on total chromium (III and VI).

Ten-day HA --  $1.4E+0$  mg/L

NOAEL -- 14.4 mg/kg/day

UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)

Assumptions -- 1 L/day water consumption for a 10-kg child

Principal Study -- Gross and Heller, 1946

Rats were exposed to drinking water containing Cr(VI) ( $K_2CrO_4$ ) at levels of 80 or 134 mg Cr(VI)/L for 60 days (8.3 or 14.4 mg Cr(VI)/kg/day, respectively) without adverse effects. Therefore, a NOAEL of 14.4 mg/kg/day is identified.

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#### HALTC-

NOTE: All chromium HAs are based on total chromium (III and VI).

Longer-term (Child) HA --  $2.4E-1$  mg/L

NOAEL -- 2.4 mg/kg/day

UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)

Assumptions -- 1 L/day water consumption for a 10-kg child

Principal study -- MacKenzie et al., 1958

In a 1-year drinking water study, consumption of water containing either Cr(III) ( $CrCl_3$ ) or Cr(VI) ( $K_2CrO_4$ ) (0 to 1.87 mg/kg/day for male rats and 0 to 2.41 mg/kg/day for female rats) produced no significant differences in weight gain, appearance, or pathological changes in the blood or other tissue. Therefore, a NOAEL of 2.41 mg/kg/day is identified.

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#### HALTA-

NOTE: All chromium HAs are based on total chromium (III and VI).

Longer-term (Adult) HA --  $8.4E-1$  mg/L

NOAEL -- 2.4 mg/kg/day

UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)

Assumptions -- 2 L/day water consumption for a 70-kg adult

Principal study -- MacKenzie et al., 1958 (study described in HALTC)

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#### HALIF-

NOTE: All chromium HAs are based on total chromium (III and VI).

Drinking Water Equivalent Level (DWEL) --  $1.7E-1$  mg/L

Assumptions -- 2 L/day water consumption for a 70-kg adult

RfD Verification Date = 02/05/86 (see RDO)

Lifetime HA -- 1.2E-1 mg/L

Assumptions -- 71% exposure by drinking water

Principal study -- MacKenzie et al., 1958 (This study was used in the derivation of the chronic oral RfD; see RDO)

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OLEP -

No data available

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ALAB -

Determination of chromium is by an atomic absorption technique using either direct aspiration into a flame or a furnace.

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TREAT-

The treatment technologies that are available to remove chromium from water include coagulation/filtration, lime softening, ion exchange, and reverse osmosis.

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HADR -

o HEALTH ADVISORY SOURCE :

U.S. EPA. 1985. Draft of the Drinking Water Criteria Document on Chromium. Office of Drinking Water, Washington, DC.  
DOCUMENT

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o HEALTH ADVISORY REVIEW :

EPA review of HAs in 1985.

Public review of HAs following notification of availability in October, 1985.

Scientific Advisory Panel review of HAs in January, 1986.

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o EPA DRINKING WATER CONTACT :

Kenneth Bailey / ODW -- (202)260-5535 / FTS 260-5535

Edward V. Ohanian / ODW -- (202)260-7571 / FTS 260-7571

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WQCHU-

Water and Fish Consumption:  $1.7E+5$  ug/L

Fish Consumption Only:  $3.433E+6$  ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The WQC of  $1.7E+5$  ug/L is based on consumption of contaminated aquatic organisms and water. A WQC of  $3.433E+6$  ug/L has also been established based on consumption of contaminated aquatic organisms alone.

Reference -- 45 FR 79318 (11/28/80); 50 FR 30784 (07/29/85)

EPA Contact -- Criteria and Standards Division / OWRS  
(202)260-1315 / FTS 260-1315

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WQCAQ-

Freshwater:

Acute --  $9.8E+2$  ug/L (hardness dependent)  
Chronic --  $1.2E+2$  ug/L (hardness dependent)

Marine:

Acute LEC --  $1.03 E+4$  ug/L  
Chronic LEC -- none

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute and chronic tests on a variety of species. For freshwater aquatic life the concentration (in ug/L) of total recoverable trivalent chromium should not exceed the numerical value given by the equations " $e^{(0.8190 [\ln (\text{hardness}) + 3.688])}$ " for acute exposure and " $e^{(0.8190 [\ln (\text{hardness}) + 1.561])}$ " for chronic exposure (\*\* indicates exponentiation; hardness is in mg/L). For example, at a hardness of 50 mg/L, the acute and chronic WQC would be 980 and 120 ug/L, respectively. The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LEC's are given when the minimum data required to derive water quality criteria are not available.

Reference -- 50 FR 30784 (07/29/85)

EPA Contact -- Criteria and Standards Division / OWRS  
(202)260-1315 / FTS 260-1315

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**MCLG -**

**Value (status) -- 0.1 mg/L [total chromium] (Final, 1991)**

**Considers technological or economic feasibility? -- NO**

**Discussion --** An MCLG of 0.1 mg/L for total chromium (Cr III and Cr VI) is based on EPA's RfD methodology for Cr VI, the more toxic chromium species. The MCLG is based upon a DWEL of 0.17 mg/L calculated from available human and animal data and an assumed drinking water contribution of 20 percent. An uncertainty factor of 500 was applied. The MCLG also falls into the safe and adequate daily dietary intake range of 50 to 200 mg/day for Cr III established by the National Research Council in the National Academy of Sciences (NAS, 1989).

**Reference -- 56 FR 3526 (01/30/91)**

**EPA Contact -- Health and Ecological Criteria Division / OST / (202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791**

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**MCL -**

**Value (status) -- 0.1 mg/L [total chromium] (Final, 1991)**

**Considers technological or economic feasibility? -- NO**

**Discussion --** The EPA has established an MCL equal to the MCLG of 0.1 mg/L.

**Monitoring requirements --** Ground water systems monitored every three years; surface water systems monitored annually; systems out of compliance must begin monitoring quarterly until system is reliably and consistently below MCL.

**Analytical methodology --** Atomic absorption/furnace technique (EPA 218.2; SM 304); inductively coupled plasma (EPA 200.7): PQL= 0.01 mg/L.

**Best available technology --** Coagulation/filtration; ion exchange; lime softening; and reverse osmosis.

**Reference -- 56 FR 3526 (01/30/91)**

**EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791**

**\_\_\_ IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMC .) for Drinking Water**

**No data available**

**\_\_\_ IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS**

No data available

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CERC -

Value (status) -- 1 pound (Statutory, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- Though "Chromium (III), insoluble salts" is not specifically designated as a CERCLA hazardous substance, insoluble chromium (III) salts would be considered hazardous substances under the CERCLA broad generic listing for "Chromium and Compounds." There is no corresponding reportable quantity (RQ) for this generic class of compounds. However, the releaser is still liable for cleanup costs if the designated Federal On-Scene Coordinator (OSC) decides to take response action with respect to the release of an insoluble chromium (III) salt that is not otherwise specifically listed as a CERCLA hazardous substance. There are two chromium (III) salts which are specifically listed as CERCLA hazardous substances, chromic acetate and chromic sulfate. Both have been assigned final RQs of 1000 pounds based on aquatic toxicity (as established under section 311(b)(4) of the Clean Water Act). Metallic chromium has been assigned a final RQ of 5000 pounds.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)260-3000 / FTS 260-3000

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RCRA -

Status -- Listed (total chromium)

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)260-3000 / FTS 260-3000

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TSCA -

No data available

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OREF - Ivankovic, S. and R. Preussmann. 1975. Absence of toxic and



carcinogenic effects after administration of high doses of chromic oxide pigment in subacute and long-term feeding experiments in rats. Food Cosmet. Toxicol. 13: 347-351.

OREF - U.S. EPA. 1984. Health Effects Assessment for Trivalent Chromium. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH. OHEA for the Office of Solid Waste and Emergency Response, Washington, DC.

IREF - None

CREF - None

HAREF- Gross, W.G., and V.G. Heller. 1946. Chromates in animal nutrition. J. Ind. Hyg. Toxicol. 28: 52-56.

HAREF- MacKenzie, R.D., R.U. Byerrum, C.F. Decker, C.A. Hoppert and R.F. Langham. 1958. Chronic toxicity studies. II. Hexavalent and trivalent chromium administered in drinking water to rats. Am. Med. Assoc. Arch. Ind. Health. 18: 232-234.

HAREF- U.S. EPA. 1985. Draft of the Drinking Water Criteria Document on Chromium. Office of Drinking Water, Washington, DC.

Option? TYPE 7/2

File 7; Entry 1; Accession No. 1144

(CAS) CAS Registry Number: 7440-47-3

(MAT) Material Name: Chromium(VI)

(SYN) Synonyms:

CHROMIC ION;  
CHROMIUM;  
CHROMIUM, ION;  
Chromium(VI);  
CHROMIUM (VI) ION

(UPD) Update Date: 03-01-91

(EFF) Effective Date: 07-01-91

(STAT) Status:

STATUS OF DATA FOR Chromium(VI)

File On-Line 03-31-87

Category (section)	Status	Last Revised
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Oral RfD Assessment (I.A.)	on-line	03-01-88
Inhalation RfC Assessment (I.B.)	pending	
Carcinogenicity Assessment (II.)	on-line	03-01-91
Drinking Water Health Advisories (III.A.)	on-line	03-01-88
U.S. EPA Regulatory Actions (IV.)	on-line	06-01-90
Supplementary Data (V.)	no data	

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(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
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No effects reported	NOAEL: 25 mg/L of chromium as K <sub>2</sub> CrO <sub>4</sub>	500	1	5E-3 mg/kg/day
Rat, 1-Year Drinking Study	(converted to 2.4 mg of chromium(VI)/kg/day)			
MacKenzie et al., 1958	LOAEL: none			
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\*Conversion Factors: Drinking water consumption = 0.097 L/kg/day (reported)

#### I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

MacKenzie, R.D., R.U. Byerrum, C.F. Decker, C.A. Hoppert and R.F. Langham.

1958. Chronic toxicity studies. II. Hexavalent and trivalent chromium administered in drinking water to rats. Am. Med. Assoc. Arch. Ind. Health. 18: 232-234.

Groups of eight male and eight female Sprague-Dawley rats were supplied with drinking water containing 0-11 ppm (0-11 mg/L) hexavalent chromium (as K<sub>2</sub>CrO<sub>4</sub>) for 1 year. The control group (10/sex) received distilled water. A second experiment involved three groups of 12 males and 9 female rats. One group was given 25 ppm (25 mg/L) chromium (as K<sub>2</sub>CrO<sub>4</sub>); a second received 25 ppm chromium in the form of chromic chloride; and the controls again received distilled water. No significant adverse effects were seen on appearance, weight gain, or food consumption, and there were no pathologic changes in the blood or other tissues in any treatment group. The rats receiving 25 ppm of chromium (as K<sub>2</sub>CrO<sub>4</sub>) showed an approximate 20% reduction in water consumption. This dose corresponds to 2.4 mg chromium(VI)/kg/day based on actual body weight and water consumption data.

For rats treated with 0-11 ppm (in the diet), blood was examined monthly, and tissues (livers, kidneys and femurs) were examined at 6 months and 1 year. Spleens were also examined at 1 year. The 25 ppm groups (and corresponding controls) were examined similarly, except that no animals were killed at 6

months. An abrupt rise in tissue chromium concentrations was noted in rats treated with greater than 5 ppm. The authors stated that "apparently, tissues can accumulate considerable quantities of chromium before pathological changes result." In the 25 ppm treatment groups, tissue concentrations of chromium were approximately 9 times higher for those treated with hexavalent chromium than for the trivalent group.

Similar no-effect levels have been observed in dogs and humans. Anwar et al. (1961) observed no significant effects in female dogs (2/dose group) given up to 11.2 ppm chromium(VI) (as  $K_2CrO_4$ ) in drinking water for 4 years. The calculated doses were 0.012-0.30 mg/kg of chromium(VI). In humans, no adverse health effects were detected (by physical examination) in a family of four persons who drank for 3 years from a private well containing chromium(VI) at approximately 1 mg/L (0.03 mg/kg/day for a 70-kg human).

#### I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF = 500. The uncertainty factor of 500 represents two 10-fold decreases in dose to account for both the expected interhuman and interspecies variability in the toxicity of the chemical in lieu of specific data, and an additional factor of 5 to compensate for the less-than-lifetime exposure duration of the principal study.

MF = 1.

#### I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

This RfD is limited to metallic chromium(VI) of soluble salts. Examples of soluble salts include potassium dichromate ( $K_2Cr_2O_7$ ), sodium dichromate ( $Na_2Cr_2O_7$ ), potassium chromate ( $K_2CrO_4$ ) and sodium chromate ( $Na_2CrO_4$ ).

Trivalent chromium is an essential nutrient. There is some evidence to indicate that hexavalent chromium is reduced in part to trivalent chromium in vivo (Petrilli and DeFlora, 1977, 1978; Gruber and Jennette, 1978).

The literature available on possible fetal damage caused by chromium compounds is limited. No studies were located on teratogenic effects resulting from ingestion of chromium.

#### I.A.5. CONFIDENCE IN THE ORAL RfD

Study: Low  
Data Base: Low  
RfD: Low

Confidence in the chosen study is low because of the small number of animals tested, the small number of parameters measured and the lack of toxic effect at the highest dose tested. Confidence in the data base is low because the supporting studies are of equally low quality, and teratogenic and reproductive endpoints are not well studied. Low confidence in the RfD follows.

#### I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

U.S. EPA. 1984. Health Effects Assessment for Hexavalent Chromium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.

U.S. EPA. 1985. Drinking Water Health Advisory for Chromium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. (Draft)

Agency RfD Work Group Review: 11/21/85, 02/05/86

Verification Date: 02/05/86

#### I.A.7. EPA CONTACTS (ORAL RfD)

Kenneth L. Bailey / ODW -- (202)382-5535 / FTS 382-5535

Christopher T. DeRosa / ORD -- (513)569-7534 / FTS 684-7534

#### I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

A risk assessment for this substance/agent is under review by an EPA work group.

(CAR) Carcinogenicity Assessment:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

#### II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- A; human carcinogen

Basis -- Results of occupational epidemiologic studies of chromium-exposed workers are consistent across investigators and study populations. Dose-response relationships have been established for chromium exposure and lung cancer. Chromium-exposed workers are exposed to both chromium III and chromium VI compounds. Because only chromium VI has been found to be carcinogenic in animal studies, however, it was concluded that only chromium VI should be classified as a human carcinogen.

#### II.A.2. HUMAN CARCINOGENICITY DATA

Sufficient. Epidemiologic studies of chromate production facilities in the United States (Machle and Gregorius, 1948; Brinton et al., 1952; Mancuso and Hueper, 1951; Mancuso, 1975; Baetjer, 1950; Taylor, 1966; Enterline, 1974; Hayes et al., 1979; Hill and Ferguson, 1979), Great Britain (Bidstrup, 1951; Bidstrup and Case, 1956; Alderson et al., 1981), Japan (Watanabe and Fukuchi, 1975; Ohsaki et al., 1978; Sano and Mitohara, 1978; Satoh et al., 1981) and West Germany (Korallus et al., 1982; Bittersohl, 1971) have established an association between chromium (Cr) exposure and lung cancer. Most of these studies did not attempt to determine whether Cr III or Cr VI compounds were the etiologic agents.

Three studies of the chrome pigment industry, one in Norway (Langard and Norseth, 1975), one in England (Davies, 1978, 1979), and the third in the Netherlands and Germany (Frentzel-Beyme, 1983) also found an association between occupational chromium exposure (predominantly to Cr VI) and lung cancer.

Results of two studies of the chromium plating industry (Royle, 1975; Silverstein et al., 1981) were inconclusive, while the findings of a Japanese study of chrome platers were negative (Okubo and Tsuchiya, 1979). The results of studies of ferrochromium workers (Pokrovskaya and Shabynina, 1973; Langard et al., 1980; Axelsson et al., 1980) were inconclusive as to lung cancer risk.

#### II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. Hexavalent chromium compounds were carcinogenic in animal

assays producing the following tumor types: intramuscular injection site tumors in Fischer 344 and Bethesda Black rats and in C57BL mice (Furst et al., 1976; Maltoni, 1974, 1976; Payne, 1960; Heuper and Payne, 1959); intraplural implant site tumors for various chromium VI compounds in Sprague-Dawley and Bethesda Black rats (Payne, 1960; Heuper 1961; Heuper and Payne, 1962); intrabronchial implantation site tumors for various Cr VI compounds in Wistar rats (Levy and Martin, 1983; Laskin et al., 1970; Levy as quoted in NIOSH, 1975); and subcutaneous injection site sarcomas in Sprague-Dawley rats (Maltoni, 1974, 1976).

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

A large number of chromium compounds have been assayed in in vitro genetic toxicology assays. In general, hexavalent chromium is mutagenic in bacterial assays whereas trivalent chromium is not (Lofroth, 1978; Petrellie and Flora, 1977, 1978). Likewise Cr VI but not Cr III was mutagenic in yeasts (Bonatti et al., 1976) and in V79 cells (Newbold et al., 1979). Chromium III and VI compounds decrease the fidelity of DNA synthesis in vitro (Loeb et al., 1977), while Cr VI compounds inhibit replicative DNA synthesis in mammalian cells (Levis et al., 1978) and produce unscheduled DNA synthesis, presumably repair synthesis, as a consequence of DNA damage (Raffetto, 1977). Chromate has been shown to transform both primary cells and cell lines (Fradkin et al., 1975; Tsuda and Kato, 1977; Casto et al., 1979). Chromosomal effects produced by treatment with chromium compounds have been reported by a number of authors; for example, both Cr VI and Cr III salts were clastogenic for cultured human leukocytes (Nakamuro et al., 1978).

There are no long-term studies of ingested Cr VI. There appears to be significant in vivo conversion of Cr VI to Cr III and III to VI; Cr III is an essential trace element.

#### II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

##### II.C.1. SUMMARY OF RISK ESTIMATES

Inhalation Unit Risk --  $1.2E-2$  per (ug/cu.m)



Extrapolation Method -- Multistage, extra risk

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
-----	-----
E-4 (1 in 10,000)	8E-3 ug/cu.m
E-5 (1 in 100,000)	8E-4 ug/cu.m
E-6 (1 in 1,000,000)	8E-5 ug/cu.m

II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION EXPOSURE

Species/Strain	Dose	Tumor	Reference
Tumor Type		Incidence	
-----		-----	
human	Route: Occupational exposure (inhalation)		
Age (years)	Midrange (ug/cu.m)	Deaths from Lung Cancer	Person Years
50	5.66	3	1345
	25.27	6	931
	46.83	6	299
60	4.68	4	1063
	20.79	5	712
	39.08	5	211
70	4.41	2	401
	21.29	4	345

II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE)

The cancer mortality in Mancuso (1975) was assumed to be due to Cr VI, which was further assumed to be no less than one-seventh of total chromium.

It was also assumed that the smoking habits of chromate workers were similar to those of the U.S. white male population. The unit risks of Langard et al. (1980), Axelsson et al. (1980), and Pokrovskaya and Shabynina (1973) are 1.3E-1, 3.5E-2 and 9.2E-2 per (ug/cu.m), respectively.

Hexavalent chromium compounds have not produced lung tumors in animals by inhalation. Trivalent chromium compounds have not been reported as carcinogenic by any route of administration.

The unit risk should not be used if the air concentration exceeds 8E-1

ug/cu.m, since above this concentration the unit risk may not be appropriate.

#### II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOSURE)

Results of studies of chromium exposure are consistent across investigators and countries. A dose-relationship for lung tumors has been established. The assumption that the ratio of Cr III to Cr VI is 6:1 may lead to a 7-fold underestimation of risk. The use of 1949 hygiene data, which may underestimate worker exposure, may result in an overestimation of risk. Further overestimation of risk may be due to the implicit assumption that the smoking habits of chromate workers were similar to those of the general white male population, since it is generally accepted that the proportion of smokers is higher for industrial workers than for the general population.

#### II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

##### II.D.1. EPA DOCUMENTATION

Mancuso, T.F. 1975. International Conference on Heavy Metals in the Environment. Toronto, Ontario, Canada.

U.S. EPA. 1984. Health Assessment Document for Chromium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. EPA 600/8-83-014F.

##### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The quantification of cancer risk in the 1984 Health Assessment Document has received peer review in public sessions of the Environmental Health Committee of the U.S. EPA's Science Advisory Board.

Agency Work Group Review: 06/26/86

Verification Date: 06/26/86

##### II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Herman J. Gibb / ORD -- (202)382-5898 / FTS 382-5898

Chao W. Chen / ORD -- (202)382-5719 / FTS 382-5719

(HA) Hazard Assessment:

III. HEALTH HAZARD ASSESSMENTS FOR VARIED EXPOSURE DURATIONS

III.A. DRINKING WATER HEALTH ADVISORIES

III.A.1. ONE-DAY HEALTH ADVISORY FOR A CHILD

Appropriate data for calculating a One-day HA are not available. It is recommended that the Ten-day HA of 1.4 mg/L be used as the One-day HA.

III.A.2. TEN-DAY HEALTH ADVISORY FOR A CHILD

Ten-day HA -- 1.4E+0 mg/L

NOAEL -- 14.4 mg/kg/day

UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)

Assumptions -- 1 L/day water consumption for a 10-kg child

Principal Study -- Gross and Heller, 1946

Rats were exposed to drinking water containing Cr(VI) ( $K_2CrO_4$ ) at levels of 80 or 134 mg Cr(VI)/L for 60 days (8.3 or 14.4 mg Cr(VI)/kg/day, respectively) without adverse effects. Therefore, a NOAEL of 14.4 mg/kg/day is identified.

III.A.3. LONGER-TERM HEALTH ADVISORY FOR A CHILD

Longer-term (Child) HA -- 2.4E-1 mg/L

NOAEL -- 2.4 mg/kg/day

UF -- 100 (allows for interspecies and intrahuman variability with the use of  
a NOAEL from an animal study)

Assumptions -- 1 L/day water consumption for a 10-kg child

Principal study -- MacKenzie et al., 1958

In a 1-year drinking water study, consumption of water containing either

Cr(III) ( $\text{CrCl}_3$ ) or Cr(VI) ( $\text{K}_2\text{CrO}_4$ ) (0 to 1.87 mg/kg/day for male rats and 0 to  
2.41 mg/kg/day for female rats) produced no significant differences in weight

gain, appearance, or pathological changes in the blood or other tissue.  
Therefore, a NOAEL of 2.41 mg/kg/day is identified.

#### III.A.4. LONGER-TERM HEALTH ADVISORY FOR AN ADULT

Longer-term (Adult) HA --  $8.4\text{E-}1$  mg/L

NOAEL -- 2.4 mg/kg/day

UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)

Assumptions -- 2 L/day water consumption for a 70-kg adult

Principal study -- MacKenzie et al., 1958 (study described in III.A.3.)

#### III.A.5. DRINKING WATER EQUIVALENT LEVEL / LIFETIME HEALTH ADVISORY

DWEL -- 1.7E-1 mg/L

Assumptions -- 2 L/day water consumption for a 70-kg adult

RfD Verification Date - 02/05/86 (see Section I.A. of this file)

Lifetime HA -- 1.2E-1 mg/L

Assumptions -- 71% exposure by drinking water

Principal study -- MacKenzie et al., 1958 (This study was used in the derivation of the chronic oral RfD; see Section I.A.2.)

#### III.A.7. ANALYTICAL METHODS FOR DETECTION IN DRINKING WATER

Determination of chromium is by an atomic absorption technique using either direct aspiration into a flame or a furnace.

#### III.A.8. WATER TREATMENT

The treatment technologies that are available to remove chromium from water include coagulation/filtration, lime softening, ion exchange, and reverse osmosis.

#### III.A.9. DOCUMENTATION AND REVIEW OF HAS

U.S. EPA. 1985. Draft of the Drinking Water Criteria Document on Chromium.

Office of Drinking Water, Washington, DC.

EPA review of HAS in 1985.

Public review of HAS following notification of availability in October, 1985.

Scientific Advisory Panel review of HAS in January, 1986.

Preparation date of this IRIS summary -- 06/22/87

#### III.A.10. EPA CONTACTS

Kenneth Bailey / ODW -- (202)382-5535 / FTS 382-5535

Edward V. Ohanian / ODW -- (202)382-7571 / FTS 382-7571

(REGS) Regulations:

IV. U.S. EPA REGULATORY ACTIONS

IV.A. CLEAN AIR ACT (CAA)

IV.A.1. CAA REGULATORY DECISION

Action -- Intent to list under Section 112

Considers technological or economic feasibility? -- NO

Discussion -- Chromium VI is considered a human carcinogen (IARC Group I), and according to EPA's preliminary risk assessment from ambient air exposures, public health risks are significant. There is considerable uncertainty as to the carcinogenicity of other valence states of chromium and the proportion of chromium VI in emission or ambient air samples. The EPA indicated that it intends to add total chromium or chromium VI to the list of hazardous air pollutants for which it intends to establish emission standards under section 112(b)(1)(A) of the Clean Air Act. The EPA will decide whether to add total chromium or chromium VI to the list only after studying possible techniques that might be used to control emissions and further assessing the public health risks. The EPA will add total chromium or chromium VI to the list if emission standards are warranted.

Reference -- 50 FR 24317 (06/10/85)

EPA Contact -- Emissions Standards Division, OAQPS  
(919)541-5571 / FTS 629-5571

IV.B. SAFE DRINKING WATER ACT (SDWA)

IV.B.1. MAXIMUM CONTAMINANT LEVEL GOAL (MCLG) for Drinking Water

Value (status) -- 0.12 mg/L [total chromium] (Proposed, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- An MCLG of 0.12 mg/L for total chromium (Cr III and Cr VI) is proposed based on a provisional DWEL of 0.17 mg/L with data on human exposure factored in (0.10 mg/day in the diet and 0 mg/day by air). A DWEL of 0.17

mg/L was calculated from a NOAEL of 2.41 mg/kg/day in rats [1-year drinking water study (Cr VI)], with an uncertainty factor of 500 applied and consumption of 2 L of water/day assumed.

Reference -- 50 FR 46936 Part IV (11/13/85)

EPA Contact -- Kenneth Bailey / Criteria and Standards Division, ODW / (202)382-7571 / FTS 382-7571; or Drinking Water Hotline / (800)426-4791

#### IV.B.2. MAXIMUM CONTAMINANT LEVEL (MCL) for Drinking Water

Value (status) -- 0.05 mg/L [total chromium] (Interim, 1980)

Considers technological or economic feasibility? -- NO

Discussion --

Reference -- 45 FR 57332

EPA Contact -- Kenneth Bailey / Criteria and Standards Division, ODW / (202)382-7571 / FTS 382-7571; or Drinking Water Hotline / (800)426-4791

#### IV.C. CLEAN WATER ACT (CWA)

##### IV.C.1. AMBIENT WATER QUALITY CRITERIA, Human Health

Water and Fish Consumption -- 5.0E+1 ug/L

Fish Consumption Only -- None

Considers technological or economic feasibility? -- NO

Discussion --

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division, OWRS  
(202)475-7315 / FTS 475-7315

##### IV.C.2. AMBIENT WATER QUALITY CRITERIA, Aquatic Organisms

###### Freshwater:

Acute -- 1.6E+1 ug/L (1-hour average)

Chronic -- 1.1E+1 ug/L (4-day average)

###### Marine:

Acute -- 1.1E+3 ug/L (1-hour average)

Chronic -- 5.0E+1 ug/L (4-day average)

Considers technological or economic feasibility? -- NO

Discussion --

Reference -- 50 FR 30784 (07/28/85)

EPA Contact -- Criteria and Standards Division, OWRS  
(202)475-7315 / FTS 475-7315

#### IV.F. RESOURCE CONSERVATION AND RECOVERY ACT (RCRA)

##### IV.F.1. RCRA APPENDIX IX, for Ground Water Monitoring

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

#### IV.G. SUPERFUND (CERCLA)

##### IV.G.1. REPORTABLE QUANTITY (RQ) for Release into the Environment

Value (status) -- 1 pound (Proposed, 1987)

Considers technological or economic feasibility? -- NO

Discussion -- The proposed RQ for chromium is based on potential carcinogenicity. Available epidemiological data on inhalation of hexavalent

chromium indicate a hazard ranking of high based on a potency factor of 388.99/mg/kg/day and assignment to weight-of-evidence group A. This corresponds to an RQ of 1 pound.

Reference -- 52 FR 8140 (03/16/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000



Option? TYPE 13/2

File 13; Entry 1; Accession No. 1455

(CAS) CAS Registry Number: 218-01-9

(MAT) Material Name: Chrysene

(SYN) Synonyms:

Chrysene;  
BENZ(a)PHENANTHRENE;  
BENZO(a)PHENANTHRENE;  
Chrysene;  
HSDB 2810;  
NSC 6175;  
RCRA WASTE NUMBER U050;  
1,2-BENZOPHENANTHRENE;  
1,2-BENZPHENANTHRENE;  
1,2,5,6-DIBENZONAPHTHALENE

(UPD) Update Date: 12-01-90

(EFF) Effective Date: 07-01-91

(STAT) Status:

STATUS OF DATA FOR Chrysene

File On-Line 12-01-90

Category (section)	Status	Last
Revised		
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Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	
12-01-90		
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	no data	
Supplementary Data (V.)	no data	

(CAR) Carcinogenicity Assessment:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen

Basis -- No human data and sufficient data from animal bioassays. Chrysene produced carcinomas and malignant lymphoma in mice after intraperitoneal injection and skin carcinomas in mice following dermal exposure. Chrysene produced chromosomal abnormalities in hamsters and mouse germ cells after gavage exposure, positive responses in bacterial gene mutation assays and transformed mammalian cells exposed in culture.

II.A.2. HUMAN CARCINOGENICITY DATA

None. Although there are no human data that specifically link exposure to chrysene to human cancers, chrysene is a component of mixtures that have been associated with human cancer. These include coal tar, soots, coke oven emissions and cigarette smoke (U.S. EPA, 1984, 1990; IARC, 1983, 1984).

II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. Intraperitoneal chrysene injections in male mice caused an increased incidence of liver tumors (Wislocki et al., 1986; Buening et al., 1979) and increased incidences of malignant lymphoma and lung tumors (Wislocki et al., 1986). In mouse skinpainting assays chrysene tested positive in both initiation and complete carcinogen studies (Wynder and Hoffman, 1959).

On days 1, 8, and 15 of age, groups of male (28 to 35/group) and female (24 to 34/group) CD-1 mice received intraperitoneal injections

of chrysene in dimethyl sulfoxide (DMSO) (total dose = 0, 160 ug or 640 ug/mouse) (Wislocki et al., 1986). The low-dose and high-dose experiments were initiated 10 weeks apart and had separate concurrent vehicle controls. Tumors were evaluated in animals that died spontaneously after weaning and in all remaining animals at 1 year after exposure. A statistically significant increase in the incidence of liver adenomas or carcinomas occurred in treated male mice relative to their respective controls: 10/35 (29%) and 5/45 (11%) in the low-dose mice and controls, respectively; and 14/34 (41%) and 2/28 (7%) in the high-dose mice and controls, respectively. The majority of the liver tumors in the high-dose males were carcinomas and the incidence was statistically significantly greater than in its respective control group, whereas the majority of tumors in the low-dose males were adenomas. Liver adenomas, but no carcinomas were observed in the control groups. In female mice no tumors were observed. The incidence of lung adenomas or carcinomas in the low-dose male mice was 6/35 (17%) (one of which was a carcinoma) and 4/45 (9%) (two of which were carcinomas) in their control group. The incidence of lung adenomas was statistically elevated in high-dose males 7/34 (21%) when compared with their control group (1/28, 4%). The incidence of malignant lymphoma was significantly elevated (3/35, 9%) in low-dose males relative to the controls (0/45), but not in the high-dose males (1/34) relative to their controls (1/28). In females, there was no statistically significant increase in lung tumors or lymphoma. This is generally regarded as a short-term exposure study with a less-than-lifetime (1 year) experiment.

Male and female Swiss Webster BLU/Ha(ICR) mice received intraperitoneal injections of chrysene in DMSO (total dose = 320 ug/mouse) or DMSO alone on days 1, 8 and 15 after birth (Buening et al., 1979). Mice were killed at 38-42 weeks of age. The incidences of lung tumors in the treated

group appeared to be elevated (5/24 (21%) and 1/11 (9%) in males and females, respectively), although not statistically significantly, when compared with the control groups (2/21 (10%) and 7/38 (18%) in males and females, respectively). The incidence of hepatic tumors in the treated males was statistically significantly greater (6/24, 25%) than in control males (0/21), whereas no hepatic tumors were found in the females. In a replication of this study, lung tumor incidence was not increased; however, the incidence of hepatic tumors in treated male mice was significantly elevated (6/27, 22%) over the incidence in the control group (0/52) (Chang et al., 1983). No liver tumors

were reported in the females. These studies are regarded as short-term exposure, less-than-lifetime experiments.

Chrysene has been tested for complete carcinogenic activity and initiating activity in mouse skin painting assays. It was shown to be a complete carcinogen (Wynder and Hoffmann, 1959). Chrysene has produced positive results for initiating activity in several mouse strains (C3H, ICR/Ha Swiss, Ha/ICR/Mil Swiss, CD-1, Sencar) when applied in combination with various promoting agents (decahydronaphthalene, croton oil, TPA) producing skin papillomas and carcinomas (Van Duuren et al., 1966; Scribner, 1973; Horton and Christian, 1974; Hecht et al., 1974; Levi et al., 1978; Wood et al., 1979, 1980; Slaga et al., 1980; Rice et al., 1985).

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Chrysene produced positive results in tests for reverse mutation in three strains of *Salmonella typhimurium* and positive results for forward mutation in one strain (McCann et al., 1975; Tokiwa et al., 1977; Wood et al., 1977; LaVoie et al., 1979; Dunkel and Simmon, 1980; Sakai et al., 1985; Kaden et al., 1979).

Chromosomal effects were observed in Chinese hamster cells, mouse oocytes and hamster spermatogonia following gavage doses of 450 or 900 mg/kg (Basler et al., 1977; Roszinsky-Kocher et al., 1979). Positive results were obtained (10 ug/mL) in tests for cell transformation in Syrian hamster embryo cells and negative results in mouse prostrate C3HG23 cells (Marquardt and Heidelberger, 1972; Pienta et al., 1977).

Current theories on mechanisms of metabolic activation of polycyclic aromatic hydrocarbons are consistent with a carcinogenic potential for chrysene. Chrysene has a "bay-region" in structure (Jerina et al., 1978). It is metabolized by mixed function oxidases to reactive "bay-region" diol epoxides (Nordqvist et al., 1981; Vyas et al., 1982) that are mutagenic in bacteria and tumorigenic in mouse skin painting assays and when injected into newborn mice (Levin et al., 1978; Wood et al., 1977, 1979; Slaga et al., 1980; Chang et al., 1983).

## II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1984. Carcinogen Assessment of Coke Oven Emissions. Office of Health and Environmental Assessment, Washington, DC. EPA 600/6-82-003F. NTIS PB 84-170181.

U.S. EPA. 1990. Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons (PAHs). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. Final Draft. ECAO-CIN-D010, September, 1990.

### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The 1990 Drinking Water Criteria Document for Polychlorinated Aromatic

Hydrocarbons has received Agency and external review.

Agency Work Group Review: 02/07/90

Verification Date: 02/07/90

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Rita S. Schoeny / ORD -- (513)569-7544 / FTS 684-7544

Robert E. McGaughy / ORD -- (202)382-5889 / FTS 382-5889

Option? TYPE 2/2

File 2; Entry 1; Accession No. 1368

(CAS) CAS Registry Number: 7440-50-8

(MAT) Material Name: Copper

(SYN) Synonyms:  
Copper

(UPD) Update Date: 09-07-88

(EFF) Effective Date: 07-01-91

(STAT) Status:  
STATUS OF DATA FOR Copper

File On-Line 09-07-88

Category (section) Revised	Status	Last
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Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.) 09-07-88	on-line	
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	no data	
Supplementary Data (V.)	no data	

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(CAR) Carcinogenicity Assessment:  
I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC  
EFFECTS  
II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

## II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

### II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classified

Basis -- There are no human data, inadequate animal data from assays of copper compounds, and equivocal mutagenicity data.

### II.A.2. HUMAN CARCINOGENICITY DATA

None.

### II.A.3. ANIMAL CARCINOGENICITY DATA

Inadequate. Bionetics Research Labs (1968) studied the carcinogenicity of a copper-containing compound, copper hydroxyquinoline, in two strains of mice (B6C3F1 and B6AKF1). Groups of 18 male and 18 female 7-day-old mice were administered 1000 mg copper hydroxyquinoline/kg bw (180.6 mg Cu/kg) suspended in 0.5% gelatin daily until they were 28 days old, after which they were administered 2800 ppm (505.6 ppm Cu) in the feed for 50 additional weeks. No statistically significant increases in tumor incidence were observed in the treated 78-week-old animals.

In the same study, Bionetics Research Labs (1968) administered a single subcutaneous injection of gelatin (control) or 1000 mg of copper hydroxyquinoline/kg bw (180.6 mg Cu/kg) suspended in 0.5% gelatin to groups of 28-day-old mice of both strains. After 50 days of observation, the male B6C3F1 had an increased incidence of reticulum cell sarcomas compared with controls. No tumors were observed in the treated male B6AKF1 mice, and a low incidence of reticulum cell sarcomas was observed in the treated female mice of both strains.

Gilman (1962) administered intramuscular injections containing 20 mg of cupric oxide (16 mg Cu), cupric sulfide (13.3 mg Cu), and cuprous sulfide (16 mg Cu) into the left and right thighs of 2- to 3-month-old Wistar rats.



After 20 months of observations, no injection-site tumors were observed in any animals, but other tumors were observed at very low incidence in the animals receiving cupric sulfide (1/30) and cuprous sulfide (1/30). As the relevance of the organic copper compound to the observation of sarcoma induction is uncertain and the incidence of tumors in rats treated i.m. with inorganic copper was very low, data are considered inadequate for classification.

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Moriya et al. (1983) reported no increase in mutations in *E. coli* and *S.*

*typhimurium* strains TA98, TA1535, TA1537 and TA1538 incubated with up to 5 mg copper quinolinolate/plate and in *S. typhimurium* TA98 and TA100 incubated

with up to 5 mg copper sulfate/plate. Demerec et al. (1951) reported

dose-related mutagenic effects in *E. coli* with 2 to 10 ppm copper sulfate in

a reverse mutation assay. Negative results were obtained with copper sulfate

or copper chloride in assays using *S. cerevisiae* (Singh, 1983) and *Bacillus*

*subtilis* (Nishioka, 1975, Matsui, 1980, Kanematsu et al., 1980). Errors in

DNA synthesis from poly(c)templates have been induced in viruses incubated

with copper chloride or copper acetate (Sirover and Loeb, 1976). Chromosomal

aberrations were induced in isolated rat hepatocytes when incubated with

copper sulfate (Sina et al., 1983). Casto et al. (1979) showed enhanced cell

transformation in Syrian hamster embryo cells infected with simian adenovirus

with the addition of cuprous sulfide and copper sulfate. High concentrations

of copper compounds have been reported to induce mitosis in rat ascites cells

and recessive lethals in *Drosophila melanogaster*. Law (1983) reported

increases in the percent lethals observed in *Drosophila* larvae and eggs when

exposed to copper by microinjection (0.1% copper sulfate) or immersion

(concentrated aqueous copper sulfate), respectively.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS  
(CARCINOGENICITY ASSESSMENT)

II.D.1. EPA DOCUMENTATION

U.S. EPA. 1987. Drinking Water Criteria Document for Copper.  
Prepared by  
the Office of Health and Environmental Assessment, Environmental  
Criteria and  
Assessment Office, Cincinnati, OH for the Office of Drinking  
Water,  
Washington, DC. ECAO-CIN 417.

Bionetics Research Labs. 1968. Evaluation of carcinogenic,  
teratogenic and  
mutagenic activities of selected pesticides and industrial  
chemicals. Vol.

I. Carcinogenic study prepared for National Cancer Institute.

NCI-DCCP-CG-1973-1-1.

Castro, B.C., J. Meyers and J.A. DiPaolo. 1979. Enhancement of  
viral  
transformation for evaluation of the carcinogenic or mutagenic  
potential of  
inorganic metal salts. Cancer Res. 30: 193.

Demerec, M., G. Bertani and J. Flint. 1951. A survey of  
chemicals for  
mutagenic action on E. coli. Am. Natur. 85: 119.

Gilman, J.P.W. 1962. Metal carcinogenesis. II. A study on the  
carcinogenic  
activity of cobalt, copper, iron and nickel compounds. Cancer  
Res. 22:  
158-166.

Kanematsu, N., M. Hara and T. Kada. 1980. Rec assay and  
mutagenicity  
studies on metal compounds. Mutat. Res. 77: 109-116.

Matsui, S. 1980. Evaluation of a Bacillus subtilis rec-assay  
for the  
detection of mutagens which may occur in water environments.  
Water Res.  
14(11): 1613-1619.

Moriya, M., T. Ohta, K. Watanabe, T. Miyazawa, K. Kato and Y.  
Shirasu.  
1983. Further mutagenicity studies on pesticides in bacterial  
reversion  
assay systems. Mutat. Res. 116(3-4): 185-216.

Nishioka, H. 1975. Mutagenic activities of metal compounds in bacteria.  
Mutat. Res. 31: 185-189.

Sina, J.F., C.L. Bean, G.R. Dysart, V.I. Taylor and M.O. Bradley. 1983.  
Evaluation of the alkaline elution/rat hepatocyte assay as a predictor of carcinogenic/mutagenic potential. Mutat. Res. 113(5): 357-391.

Singh, I. 1983. Induction of reverse mutation and mitotic gene conversion by some metal compounds in *Saccharomyces cerevisiae*. Mutat. Res. 117(1-2): 149-152.

Sirover, M.A. and L.A. Loeb. 1976. Infidelity of DNA synthesis in vitro: Screening for potential metal mutagens or carcinogens. Science. 194: 1434-1436.

#### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The values in the 1987 Drinking Water Criteria Document for Copper have received peer and administrative review.

Agency Work Group Review: 09/15/87

Verification Date: 09/15/87

#### II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

David J. Reisman / ORD -- (513)569-7588 / FTS 684-7588

W. Bruce Peirano / ORD -- (513)569-7540 / FTS 684-7540

(CAS) CAS Registry Number: 53-70-3

(MAT) Material Name: Dibenz[a,h]anthracene

(SYN) Synonyms:

Dibenz(a,h)anthracene;  
DB(a,h)A;  
DBA;  
dibenz(a,h)anthracene;  
DIBENZO(a,h)ANTHRACENE;  
HSDB 5097;  
NSC 22433;  
RCRA WASTE NUMBER U063;  
1,2,5,6-DIBENZANTHRACEEN [Dutch];  
1,2,5,6-dibenzanthracene;  
1,2:5,6-BENZANTHRACENE;  
1,2:5,6-DIBENZ(a)ANTHRACENE;  
1,2:5,6-Dibenzanthracene;  
1,2:5,6-DIBENZOANTHRACENE

(UPD) Update Date: 12-01-90

(EFF) Effective Date: 10-01-91

(STAT) Status:

STATUS OF DATA FOR Dibenz[a,h]anthracene

File On-Line 12-01-90

Category (section)	Status	Last Revised
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Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	12-01-90
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	no data	
Supplementary Data (V.)	no data	

(CAR) Carcinogenicity Assessment:

- I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS
- II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE
  - II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY
    - II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen

Basis -- Based on no human data and sufficient data from animal bioassays. Dibenz[a,h]anthracene produced carcinomas in mice following oral or dermal exposure and injection site tumors in several species following subcutaneous or intramuscular administration. Dibenz[a,h]anthracene has induced DNA damage and gene mutations in bacteria as well as gene mutations and transformation in several types of mammalian cell cultures.

#### II.A.2. HUMAN CARCINOGENICITY DATA

None. Although there are no human data that specifically link exposure to dibenz[a,h]anthracene with human cancers, dibenz[a]anthracene is a component of mixtures that have been associated with human cancer. These include coal tar, soots, coke oven emissions and cigarette smoke (U.S. EPA, 1984, 1990; IARC, 1984).

#### II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. Dibenz[a,h]anthracene has been shown to be carcinogenic when administered to mice by the oral route (Snell and Stewart, 1962, 1963). Instead of drinking water DBA/2 mice (21/sex) were given a water-olive oil emulsion containing 0.2 mg/mL dibenz[a,h]anthracene ad libitum. Average exposure was estimated to be 0.85 mg/day for males and 0.76 mg/day for females. The control groups (25 male and 10 female) received the water-olive oil emulsion in place of water. The mice did not tolerate the olive oil vehicle well and all 4 groups lost weight after a few weeks exposure and eventually became emaciated and dehydrated. Animals that died spontaneously or that became moribund were examined for tumors. The duration of the experiment was 279 and 237 days for males and females, respectively, in the dosed groups and 351 and 226 days for male and female controls. Mice developed pulmonary adenomas (treated males, 14/14; control males 1/23; treated females, 13/13; control females, 0/6), pulmonary carcinomas (treated males, 14/14; control males, 0/23; treated females, 10/13; control females, 0/6), mammary carcinoma (treated females, 12/13; control females, 0/6) and hemangioendothelioma (treated males, 10/14; control males, 0/23; treated females, 6/13; control females, 0/6). No statistical analyses appear to have been performed.

Mammary carcinomas were observed in two strains of female mice following gavage with dibenz[a,h]anthracene (Biancifiori and Caschera, 1962; Berenblum and Haran, 1955). Biancifiori and Caschera (1962) observed mammary carcinomas when female Balb/c (1/20) and pseudo-pregnant female (obtained by mating virgin females with vasectomized males) Balb/c (13/24) mice were treated for 15 weeks with a twice-weekly gavage containing 0.5% dibenz[a,h]anthracene (total dose was 15 mg/animal). Mammary carcinomas occurred in 2/30 pseudo-pregnant females not dosed with dibenz[a,h]anthracene. Previous studies indicated that mammary carcinomas did not occur in virgin Balb/c females (Biancifiori et al., 1959). A single 1.5-mg dose of dibenz[a,h]anthracene in polyethylene glycol [average molecular weight (a.m.u.) 400] (PEG-400) produced forestomach papillomas in 2/42 male Swiss mice after 30 weeks. In this short-term study no mice developed tumors when treated with PEG alone (1 time/week) for 30 weeks (0/20) (Berenblum and Haran, 1955).

Dibenz[a,h]anthracene has produced positive results in mouse skin painting assays for complete carcinogenicity. Swiss mice developed carcinomas following dermal exposure to dibenz[a,h]anthracene at concentrations of 0.001% or greater (Wynder and Hoffman, 1959; Van Duuren et al., 1967). Numerous studies that demonstrate complete carcinogenic activity and initiating activity are summarized in IARC (1973) and U.S. EPA (1990).

Subcutaneous injection of dibenz[a,h]anthracene induced sarcomas at the site of injection in several animal species. Groups (>19) of C3H mice received single subcutaneous injections of dibenz[a,h]anthracene in tricaprillin at doses ranging from 0.0019-8 mg (approximately 0.09-360 mg/kg). No controls appear to have been used in this experiment (Bryan and Shimkin, 1943). Tumor latency appeared to decrease and the incidence of injection site sarcomas appeared to increase with dose (>76% at doses >0.06 mg or 2.8 mg/kg). A single subcutaneous injection of 2.4, 4.7, 9.3, 18.7, 37.5, or 75 ug dibenz[a,h]anthracene into groups of 100 NMRI mice was reported to produce a dose-related increase in tumor incidence (37/100, 39/100, 44/100, 56/100, 65/100, and 69/100, respectively) by the 114th week after injection (Pfeiffer, 1977). No concurrent controls were reported; however, a spontaneous tumor rate for NMRI mice was previously reported to be 0-2% (Pfeiffer, 1973). The development of fibrosarcomas from a single subcutaneous injection of 150 ug dibenz[a,h]anthracene was shown to be higher in AHH+ strains of mice than in AHH- strains.

Lubet et al. (1983) found that subcutaneous injections of dibenz[a,h]anthracene were associated with fibrosarcoma development in mice, but only for some strains. Four strains of mice used included two, C3H/HeJ and C57Bl/6J, that respond to 3-methylcholanthrene treatment with increased levels and types of hepatic enzymes, including AHH. Two strains, AKR/J and DBA/2J were nonresponders. Groups of 30 animals were injected with a single dose of 150 mg dibenz[a,h]anthracene in 0.05 mL trioctanoin and observed for 9 months. A control group for each strain, consisting of 10 animals each, received a subcutaneous injection of 0.05 mL trioctanoin alone. The tumor incidence in the treated animals varied between 0 and 80%, depending on the strain. Tumor incidences were higher in the C3H and C57Bl mice but not in AKR or DBA mice. Likewise, the average latency period (in days) for fibrosarcoma development varied with the strain and tended to be inversely correlated with the tumor incidence rate. Numerous earlier studies that demonstrate the carcinogenicity of parenterally injected dibenz[a,h]anthracene in a variety of species are summarized in IARC (1973) and U.S. EPA (1990).

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Dibenz[a,h]anthracene has produced positive results in bacterial DNA damage and mutagenicity assays and in mammalian cell DNA damage, mutagenicity and cell transformation assays. In bacterial DNA damage assays, positive results were obtained in *Escherichia coli* and *Bacillus subtilis* at exposure levels of 12-50 ug/well. Dibenz[a,h]anthracene tested positive for reverse mutation in *Salmonella typhimurium* strains TA100 and TA98 (3-5 ug/plate) and positive for forward mutation in strain TM677 (21 ug/mL) (McCann et al., 1975; Andrews et al., 1978; Baker et al., 1980; Hermann, 1981; Kaden et al., 1979). In mammalian cell DNA damage assays, positive results were obtained in human foreskin epithelial cells not activated with mixed-function oxidase (MFO) inducers (1-100 ug/mL) and in HeLa cells (28 ng/mL) activated with 3-

methylcholanthrene (Lake et al., 1978; Martin et al., 1978). When Syrian hamster embryo cells and rat hepatocytes not activated with MFO inducers were exposed to 20-30 ug/mL the results were not positive (Casto, 1979; Probst et al., 1981). Dibenz[a,h]anthracene induced forward mutations in Chinese hamster embryo cells exposed to concentrations of 1 ug/mL or greater (Huberman and Sachs, 1976; Krahn and Heidelberger, 1977; Huberman, 1978). It transformed several types of mammalian cells exposed to concentrations of 10 ug/mL or greater; these cell types included: Syrian hamster embryo cells, mouse C3H10T 1/2 cells and mouse prostate C3H cells (DiPaolo et al., 1969; Chen and Heidelberger, 1969; Pienta et al., 1977; Casto et al., 1977; Casto, 1979; Reznikoff et al., 1973; Lubet et al., 1983).

Current theories on mechanisms of metabolic activation of polycyclic aromatic hydrocarbons are consistent with a carcinogenic potential for dibenz[a,h]anthracene. Dibenz[a,h]anthracene has a "bay-region" structure (Jerina et al., 1978). It is metabolized by mixed-function oxidases to dihydrodiols that are mutagenic in bacteria and tumorigenic in mouse skin painting assays and when injected into newborn mice (Wood et al., 1978; Nordqvist et al., 1979; Slaga et al., 1980; Buening et al., 1979).

#### II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

##### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1984. Carcinogen Assessment of Coke Oven Emissions. Office of Health and Environmental Assessment, Washington, DC. EPA 600/6-82-003F. NTIS PB 84-170181.

U.S. EPA. 1990. Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons (PAHs). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. Final Draft. ECAO-CIN-D010, September, 1990.

##### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The 1990 Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons has received Agency and external review.

Agency Work Group Review: 02/07/90

Verification Date: 02/07/90

##### II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Rita S. Schoeny / ORD -- (513)569-7813 / FTS 684-7813

Robert E. McGaughy / ORD -- (202)260-5889 / FTS 260-5889

(CAS) CAS Registry Number: 132-64-9

(MAT) Material Name: Dibenzofuran

(SYN) Synonyms:  
 (1,1'-BIPHENYL)-2,2'-DIYL OXIDE;  
 2,2'-BIPHENYLENE OXIDE;  
 2,2'-BIPHENYLYLENE OXIDE;  
 DIBENZOFURAN;  
 DIBENZO(B,D)FURAN;  
 DIPHENYLENE OXIDE;  
 HSDB 2163;  
 NSC 1245

(UPD) Update Date: 10-01-90

(EFF) Effective Date: 10-01-91

(STAT) Status:  
 STATUS OF DATA FOR Dibenzofuran

File On-Line 10-01-90

Category (section)	Status	Last Revised
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Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	10-01-90
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	no data	
Supplementary Data (V.)	no data	

(CAR) Carcinogenicity Assessment:  
 I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS  
 II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE  
 II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY  
 II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity



Basis -- Based on no human data and no animal data for dibenzofuran alone.

#### II.A.2. HUMAN CARCINOGENICITY DATA

None. There are no data on the possible carcinogenicity of dibenzofuran alone in humans. Studies have evaluated exposure to a mixture of polychlorinated biphenyls (PCBs), polychlorinated dibenzofurans (PCDFs) and polychlorinated quinones (PCQs) by consumption of contaminated rice oil (Yusho incident) (reviewed in U.S. EPA, 1986, 1987). However, these studies have limited value because they do not assess dibenzofuran or correlate exposure with cancer risk. Additionally, because of the multiple exposures, the extent to which the various components contributed to the increase in cancer mortality cannot be determined.

#### II.A.3. ANIMAL CARCINOGENICITY DATA

None. No animal carcinogenicity data on dibenzofuran are currently available. U.S. EPA (1986) noted that the biological activity of PCDFs varies greatly, so that risk assessment of dibenzofuran by analogy to any of these more widely studied compounds would not be recommended.

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Dibenzofuran is not mutagenic with or without metabolic activation in several strains of *Salmonella typhimurium* assay (Schoeny, 1982).

In a comparison of Toxic Equivalency Factor (TEF) values for chlorinated dibenzofurans, mono-, di- and tri-chlorinated dibenzofuran had TEF values of 0 (U.S. EPA, 1989). Based on these results and the fact that toxicity of polychlorinated dibenzofurans (PCDF) depends on the number of chlorine substituents and their position (U.S. EPA, 1986), the TEF for dibenzofuran, with no chlorine substituents, is set equal to 0.

#### II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

None.

#### II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

None.

#### II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

##### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1986. Health Assessment Document for Polychlorinated Dibenzofurans. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA 600/8-86/018A. NTIS PB86-221256/AS.

U.S. EPA. 1987. Health Effects Assessment for Dibenzofuran. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Emergency and Remedial Response, Washington, DC. ECAO-CIN-H088.

U.S. EPA. 1989. Interim Procedures for Estimating Risks Associated with Exposures to Mixtures of Chlorinated Dibenzo-p-Dioxins and -Dibenzofurans (CDDs and CDFs) and 1989 Update. Risk Assessment Forum, Washington, DC. EPA/625/3-89/016.

#### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The 1986 Health Assessment for Polychlorinated Dibenzofurans is an external draft for review purposes only and does not constitute Agency policy.

The 1987 Health Effects Assessment Document for Dibenzofuran has been reviewed in accordance with the U.S. Environmental Protection Agency's peer and administrative review policies and has been approved for publication.

Agency Work Group Review: 10/05/89

Verification Date: 10/05/89

#### II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Charles Ris / ORD -- (202)260-5898 / FTS 260-5898

Rita Schoeny / ORD -- (513)569-7544 / FTS 684-7544

Captured 8/12/92

1 - IRIS  
IRSN - 36  
DATE - 920120  
UPDT - 01/20/92, 52 fields  
STAT - Oral RfD Assessment (RDO) on-line 08/01/90  
STAT - Inhalation RfC Assessment (RDI) message 10/01/90  
STAT - Carcinogenicity Assessment (CAR) on-line 08/01/91  
STAT - Drinking Water Health Advisories (DWHA) no data  
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92  
IRH - 09/07/88 CAR Carcinogen summary on-line  
IRH - 08/01/89 REFS Bibliography on-line  
IRH - 03/01/90 RDO Text corrected  
IRH - 05/01/90 CAREV First sentence revised  
IRH - 08/01/90 RDO Oral RfD summary noted as pending change  
IRH - 09/01/90 RDI Not verified; data inadequate  
IRH - 09/01/90 RCRA EPA contact changed  
IRH - 10/01/90 RDI Inhalation RfC message on-line  
IRH - 10/01/90 IREF Inhalation RfC references added  
IRH - 08/01/91 CARDR Primary and secondary contacts changed  
IRH - 01/01/92 RDO Secondary contact changed  
IRH - 01/01/92 EXSR Regulatory actions updated  
RLEN - 13854  
NAME - Dibutyl phthalate  
RN - 84-74-2  
SY - 1,2-Benzenedicarboxylic Acid Dibutyl Ester  
SY - o-Benzenedicarboxylic Acid, Dibutyl Ester  
SY - Benzene-o-Dicarboxylic Acid Di-n-Butyl Ester  
SY - Butylphthalate  
SY - Celluflex DPB  
SY - Dibutyl 1,2-Benzene dicarboxylate  
SY - Dibutyl phthalate  
SY - Di-n-Butylphthalate  
SY - Dibutyl-o-Phthalate  
SY - DPB  
SY - Elaol  
SY - Ergoplast FDB  
SY - Genoplast B  
SY - Hexaplast M/B  
SY - N-Butylphthalate  
SY - Palatinol C  
SY - Phthalic Acid Dibutyl Ester  
SY - Polycizer DBP  
SY - PX 104  
SY - RC Plasticizer DBP  
MF - C16H22O4  
USE - Plasticizer in nitrocellulose lacquers, elastomers, explosives, nail polish, and solid rocket propellants; solvent for perfume oils; perfume fixative; textile lubricating agent; safety glass; insecticides; printing inks; resin solvent; paper coatings; adhesives; insect repellants for textiles (Hawley, 1981, p. 330). Not registered as a pesticide in the U.S. (USEPA/Pesticide Index, 1985).  
COFO - Colorless, oily liquid with a weak aromatic odor (NIOSH/OSHA, 1978, p. 80)  
ODOR - Colorless, oily liquid with a weak aromatic odor (NIOSH/OSHA, 1978, p. 80)  
BP - 644F, 340C  
MP - -31F, -35C  
MW - 278.34  
DEN - 1.0484 at 20C/20C  
VAP - 1.1 at 150C  
VAPD - 9.58  
EVAP - Not Found  
SOLV - 13 mg/L at 25C  
FLPT - 315F, 157C (CC); 339.8F, 171.1C (OC)  
FLMT - Flammable Limits: LEL -- 0.5% at 456F (235C) UEL -- Not Found  
AVOI - Liquid chlorine reacts explosively with dibutyl phthalate (NFPA, 1978). Avoid contact with nitrates, strong oxidizers, strong alkalies, strong acids (NIOSH/OSHA, 1978, p. 80) and chlorine (Sax, 1984, p. 926).  
DCMP - None (NFPA, 1978)  
.....

RDO -

o ORAL RFD SUMMARY :

NOTE: The Oral Rfd for dibutyl phthalate may change in the near future pending the outcome of a further review now being conducted by the Oral Rfd Work Group.

Critical Effect	Experimental Doses*	UF	MF	Rfd
Increased mortality	NOAEL: 0.25% of diet (125 mg/kg/day)	1000	1	1E-1 mg/kg/day
Rat Subchronic to Chronic, Oral Bio- assay	LOAEL: 1.25% of diet (600 mg/kg bw/day)			

Smith, 1953

\*Conversion Factors: The values of 125 mg/kg/day for 0.25% dibutyl phthalate in the diet and 600 mg/kg/day for 1.25% were estimated from a figure depicting daily intake in mg/kg in Smith (1953).

o ORAL RFD STUDIES :

Smith, C.C. 1953. Toxicity of butyl stearate, dibutyl sebacate, dibutyl phthalate and methoxyethyl oleate. Arch. Hyg. Occup. Med. 7: 310-318.

Male Sprague-Dawley rats in groups of 10 were fed diets containing 0, 0.01, 0.05, 0.25, and 1.25% dibutyl phthalate for a period of 1 year. One-half of all rats receiving the highest dibutyl phthalate concentration died during the first week of exposure. The remaining animals survived the study with no apparent ill effects. There was no effect of treatment on gross pathology or hematology. While it was stated that several organs were sectioned and stained, no histopathologic evaluation was reported.

o ORAL RFD UNCERTAINTY :

UF = 1000. A factor of 10 was applied to account for interspecies variation, a factor of 10 for protection of sensitive human subpopulations, and an additional factor of 10 to account for both the less-than-chronic duration of the study and deficiencies in the study, such as the use of only male animals.

o ORAL RFD MODIFYING FACTOR :

MF = 1.

o ORAL RFD COMMENTS :

Fetotoxicity was observed when mice were fed 2100 mg/kg/day dibutyl phthalate throughout gestation (Shiota and Nishimura, 1982). An increase in terata of borderline statistical significance was observed in progeny of this treatment group. Dibutyl phthalate produces degeneration of the seminiferous tubules, probably as a result of increased urinary excretion of zinc (Gangolli, 1982).

o ORAL RFD CONFIDENCE :

Study: Low  
Data Base: Low  
Rfd: Low

The study by Smith (1953) used few animals of one sex only. It was not indicated in the paper whether the 50% mortality observed early in the study was considered treatment-related, nor was the cause of death indicated. This is the only subchronic bioassay of dibutyl phthalate reported in the literature. Confidence in the study, data base, and Rfd are all rated low.

o ORAL RFD SOURCE DOCUMENT :

U.S. EPA. 1980. Ambient Water Quality Criteria for Phthalate Esters. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water

Regulations and Standards, Washington, DC. EPA 440/5-80-067. NTIS PB 81-117780.

The RfD in the 1980 Ambient Water Quality Criteria document received extensive peer and public review.

o REVIEW DATES : 01/22/86  
o VERIFICATION DATE : 01/22/86  
o EPA CONTACTS :

Michael L. Dourson / ORD -- (513)569-7544 / FTS 684-7544

Adib Tabri / ORD -- (513)569-7553 / FTS 684-7553

RD1 -

o INHALATION RFD SUMMARY :

The health effects data for dibutyl phthalate were reviewed by the U.S. EPA RfD/RfC Work Group and determined to be inadequate for derivation of an inhalation RfC. The verification status of this chemical is currently not verifiable. For additional information on health effects of this chemical interested parties are referred to the EPA documentation listed below.

U.S. EPA. 1987. Drinking Water Criteria Document for Phthalic Acid Esters Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. (External Review Draft)

o REVIEW DATES : 07/26/90  
CAREV-  
o CLASSIFICATION : D; not classifiable.  
o BASIS FOR CLASSIFICATION : Pertinent data regarding carcinogenicity was not located in the available literature.  
o HUMAN CARCINOGENICITY DATA :

None.

o ANIMAL CARCINOGENICITY DATA :

None.

o SUPPORTING DATA :

DBP did not induce mutations in a modified reverse mutation plate incorporation assay in Salmonella strains TA100 and TA98 at concentrations up to 1000 ug/plate in the presence or the absence of S9 hepatic homogenate (Kozumbo et al., 1982). It was a weak direct-acting mutagen in a forward mutation assay in Salmonella typhimurium (Seed, 1982). DBP was mutagenic in the mouse lymphoma forward mutation assay only in the presence of metabolic activation (CHA, 1986). In addition, DBP showed some evidence of clastogenic activity in Chinese hamster fibroblasts (Ishidate and Odashima, 1977) but was negative in human leukocytes (Tsuchiya and Mattori, 1977). Research indicates that DBP is hydrolyzed to monoesters (Kluwe, 1982; Rowland et al., 1977; Albro and Moore, 1974). There is evidence that DBP induces peroxisome proliferation (U.S. EPA, 1987).

CARDR-

o CARCINOGENICITY SOURCE :

U.S. EPA. 1987. Drinking Water Criteria Document for Phthalic Acid Esters. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. External Review Draft.

The Drinking Water Criteria Document for Phthalic Acid Esters has received OHEA review.  
DOCUMENT

.....  
o REVIEW DATES : 08/26/87  
o VERIFICATION DATE : 08/26/87  
o EPA CONTACTS :

Welford C. Roberts / ODW -- (202)260-7589 / FTS 260-7589

Lynn Papa / ORD -- (513)569-7523 / FTS 684-7523  
.....

ACUTE-

o ACUTE TOXICITY :

Dibutyl phthalate is generally non-irritating to humans (Martin and Worthing, 1974).  
.....

o SIGNS AND SYMPTOMS :

Eye irritation with profuse tearing.  
Contact with surface of eye has caused severe stinging pain with profuse tearing (Grant, 1974). Mild throat irritation has been observed (Lefaux, 1968). Ingestion has caused nausea, dizziness, photophobia, lachrymation, and conjunctivitis (ACGIH, 1980a).  
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WQCMU-

Water and Fish Consumption:  $3.4E+4$  ug/L

Fish Consumption Only:  $1.54E+5$  ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The WQC of  $3.4E+4$  ug/L is based on consumption of contaminated aquatic organisms and water. A WQC of  $1.54E+5$  ug/L has also been established based on consumption of contaminated aquatic organisms alone.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OURS  
(202)260-1315 / FTS 260-1315  
.....

WQCAQ-

Freshwater:

Acute LEC --  $9.4E+2$  ug/L  
Chronic LEC --  $3.0E+0$  ug/L

Marine:

Acute LEC --  $2.9E+3$  ug/L  
Chronic LEC -- None

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LECs are given when the minimum data required to derive water quality criteria are not available. The values given are for the general class of phthalate esters and not specifically for dibutyl phthalate.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OURS  
(202)260-1315 / FTS 260-1315

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-----  
MCLG -

Value -- 0.8 mg/L (Proposed, 1990)

Considers technological or economic feasibility? -- NO

Discussion -- EPA is proposing to regulate dibutyl phthalate based on its potential adverse effects (increased mortality) reported in a one-year study in rats. The MCLG is based upon a DMEL of 4 mg/L and an assumed drinking water contribution of 20 percent.

Reference -- 55 FR 30370 (07/25/90)

EPA Contact -- Health and Ecological Criteria Division / OST /  
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791  
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MCL -

No data available

\_\_\_IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

\_\_\_IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

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FISTD-

Status -- List "C" Pesticide (1989)

Reference -- 54 FR 30846 (07/24/89)

EPA Contact -- Registration Branch / OPP  
(703)557-7760 / FTS 557-7760  
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FIREV-

No data available

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CERC -

Value (status) -- 10 pounds (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ is based on aquatic toxicity. The available data indicate that the aquatic 96-Hour Median Threshold Limit for dibutyl phthalate is between 0.1 and 1 ppm.

Reference -- 50 FR 13456 (04/04/85); 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)260-3000 / FTS 260-3000

.....  
RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)260-3000 / FTS 260-3000

.....  
TSCA -

No data available

- .....
- OREF - Gangolli, S.D. 1982. Testicular effects of phthalate esters. Environ. Health Perspect. 45: 77-84.
- OREF - Shiota, K. and H. Nishimura. 1982. Teratogenicity of di-2-ethylhexyl phthalate and di-n-butyl phthalate in mice. Environ. Health Perspect. 45(0): 65-70.
- OREF - Smith C.C. 1953. Toxicity of butyl stearate, dibutyl sebacate, dibutyl phthalate and methoxyethyl oleate. Arch. Hyg. Occup. Med. 7: 310-318.
- OREF - U.S. EPA. 1980. Ambient Water Quality Criteria for Phthalate Esters. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-067. NTIS PB 81- 117780.
- IREF - U.S. EPA. 1987. Drinking Water Criteria Document for Phthalic Acid Esters Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. (External Review Draft)
- CREF - Albro, P.W. and B. Moore. 1974. Identification of the metabolites of simple phthalate diesters in rat urine. J. Chromatogr. 94: 209-218.
- CREF - CMA (Chemical Manufacturers Association). 1986. Mutagenicity of 1C (di-n-butyl phthalate) in a mouse lymphoma mutation assay. Final report. Submitted to Hazleton Biotechnologies Company. MS Project No. 20989. September, 1986.
- CREF - Ishidate, M., Jr. and S. Odashima. 1977. Chromosome tests with 134 compounds on Chinese hamster cells in vitro -- A screening test for chemical carcinogens. Mutat. Res. 48: 337-354.
- CREF - Kluwe, W.M. 1982. Overview of phthalate ester pharmacokinetics in mammalian species. Environ. Health Perspect. 45: 3-10.
- CREF - Kozumbo, W.J., R. Kroll and R.J. Rubin. 1982. Assessment of the mutagenicity of phthalate esters. Environ. Health Perspect. 45: 103-109.
- CREF - Rowland, I.R., R.C. Cottrell and J.C. Phillips. 1977. Hydrolysis of phthalate esters by the gastro-intestinal contents of the rat. Food Cosmet. Toxicol. 15: 17-21.
- CREF - Seed, J.L. 1982. Mutagenic activity of phthalate esters in bacterial liquid suspension assays. Environ. Health Perspect. 45: 111-114.
- CREF - Tsuchiya, K. and K. Hattori. 1977. Chromosomal study on human leukocyte cultures treated with phthalic acid ester. Nekkaidoritus Eisei Kenkyusho No. 26: 114. (Abstract)
- CREF - U.S. EPA. 1987. Drinking Water Criteria Document for Phthalic Acid Esters. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. External Review Draft.
- NAREF- None



(CAS) CAS Registry Number: 84-66-2

(MAT) Material Name: Diethyl phthalate

(SYN) Synonyms:

ANOZOL;  
1,2-BENZENEDICARBOXYLIC ACID, DIETHYL ESTER;  
Diethyl phthalate;  
DPX-F5384;  
ESTOL 1550;  
ETHYL PHTHALATE;  
NCI-C60048;  
NEANTINE;  
PALATINOL A;  
PHTHALOL;  
PHTHALSAEUREDIAETHYLESTER;  
PLACIDOL E;  
RCRA WASTE NUMBER U088

(UPD) Update Date: 08-01-91

(EFF) Effective Date: 10-01-91

(STAT) Status:

STATUS OF DATA FOR Diethyl phthalate

File On-Line 09-30-87

Category (section)	Status	Last Revised
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Oral RfD Assessment (I.A.)	on-line	08-01-91
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	08-01-91
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	on-line	08-01-91
Supplementary Data (V.)	no data	

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(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

### I.A.1. ORAL RFD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Decreased growth rate, food consumption and altered organ weights	NOAEL: 1% of diet (750 mg/kg bw/day)  LOAEL: 5% of diet (3160 mg/kg bw/day)	1000	1	8E-1 mg/kg/day
Rat, Subchronic Oral Feeding Study Brown et al., 1978				

\*Conversion Factors: Converted doses estimated by principal study authors, based on food consumption and body weight data.

### I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RFD)

Brown, D., K.R. Butterworth, I.F. Gaunt, P. Grasso and S.D. Gangolli. 1978. Short-term oral toxicity study of diethyl phthalate in the rat. Food Cosmet. Toxicol. 16: 415-422.

Groups of CD rats (15/sex) were fed diets containing 0, 0.2, 1.0, or 5.0% DEP for 16 weeks. The authors estimated the mean intakes to be 0, 150, 770, and 3160 mg/kg/day for the males and 0, 150, 750, and 3710 mg/kg/day for the females. Additional groups of five rats/sex were fed similar diets for 2 or 6 weeks. Hematological examinations (red blood cell count, hematocrit, hemoglobin) were performed on animals fed diets for 2, 6, and 16 weeks. Differential white blood cell counts were also conducted on 0 and 5% dose groups at 16 weeks. Food and water intake and body weight were measured for all groups weekly. Urinalyses were conducted during weeks 2, 6, and 15 on 5 to 15 rats/sex/dose group. After 16 weeks of treatment, autopsy, hematology and histologic examinations were conducted on all animals.

No changes in behavior or other clinical signs of toxicity were observed. The authors reported significantly less weight gain throughout the duration of the experiment in both sexes given 5% DEP (15 to 25% decrease) and in females (5 to 8% decrease) fed 1% DEP. Mean food consumption of the previous groups was also decreased (by 11 to 23%) relative to controls. No significant dose- or time-related trends in urinalysis or hematology results were found. Absolute weights of brain, heart, spleen, and kidneys were decreased in both sexes fed 5% DEP. Relative weights of the brain, liver, kidneys, stomach, small intestines, and full caecum were significantly greater in both sexes after 16 weeks at the 5% dietary level when compared with controls. No histologic changes because of treatment were reported.

In another experiment summarized by Brown et al. (1978), groups of six rats/sex were pair-fed diets containing either 0 or 5% DEP for 16 weeks. Body weights were measured weekly. The authors reported that rats fed 5% DEP consumed more food and gained less weight than controls. The differences in food consumption (1 to 5%) were not statistically significant, and mean weight differences were 7 to 10%, which the authors reported as statistically

significant.

The RfD receives support from the results of a 2-year feeding study using rats (Food Research Laboratories, Inc., 1955). Albino weanling rats (strain not specified) (15/sex) were fed 0, 0.5, 2.5, and 5.0% diethyl phthalate in the diet. Animals were maintained for a 2-year period during which two males and two females/group were examined at 12-week intervals for the following: red and white blood cell counts, differential white count, hemoglobin, blood sugar and nitrogen, and urinalysis. Growth of animals in the 5% treatment group was retarded throughout the study, with no depression of food intake. There was a significant decrease in efficiency of food utilization in this group compared with controls. There were no other treatment-related effects either on the parameters listed above or on gross organ appearance or histopathology.

#### I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF = 1000. A factor of 10 for extrapolation from subchronic to chronic exposure, 10 for interspecies variation, and an additional 10-fold factor to protect sensitive human subpopulations were used in determining the RfD.

MF = 1.

#### I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

Data regarding developmental and reproductive effects is extremely limited. Singh et al. (1972) observed skeletal malformations in Sprague-Dawley rats after i.p. administration (0.506, 1.012, and 1.686 mL/kg) on days 5, 10, and 15 of gestation. In addition, fetuses were significantly smaller than untreated controls. Exposure to DEP does not appear to affect the reproductive performance of mice after oral administration of 0.25, 1.25, and 2.5% DEP for 18 weeks (NTP, 1984). Second-generation breeding pairs exposed to 2.5% DEP exhibited increased right epididymis and prostate weights in males and decreased pituitary weight in females (NTP, 1984).

#### I.A.5. CONFIDENCE IN THE ORAL RfD

Study: Medium  
Data Base: Low  
RfD: Low

Sufficient numbers of rats of both sexes were employed and multiple endpoints, including histopathology, were studied; confidence in the study is rated medium. Since only limited supporting data are available and the chosen study was of less than lifetime duration, confidence in the data base is rated low. Low confidence in the RfD follows.

#### I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

The RfD Work Group meeting notes of 01/22/86 directed a review of the Brown et al. (1978) study. The review has resulted in a different evaluation than presented on 01/22/86.

Agency Work Group Review: 01/22/86, 07/16/87

Verification Date: 07/16/87

**I.A.7. EPA CONTACTS (ORAL RFD)**

Welford C. Roberts / ODW -- (202)260-7589 / FTS 260-7589

Lynn Papa / ORD -- (513)569-7523 / FTS 684-7523

**(CAR) Carcinogenicity Assessment:**

**II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE**

**II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY**

**II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION**

Classification -- D; not classifiable as a human carcinogen

Basis -- Pertinent data regarding carcinogenicity were not located in the available literature.

**II.A.2. HUMAN CARCINOGENICITY DATA**

None.

**II.A.3. ANIMAL CARCINOGENICITY DATA**

Inadequate. Dietary studies in rats with exposure durations of 2 years (Food Research Laboratories, Inc., 1955) and 16 weeks (Brown et al., 1978) were not designed to measure carcinogenic effects.

**II.A.4. SUPPORTING DATA FOR CARCINOGENICITY**

DEP was found to be a weak direct-acting mutagen in forward and reverse mutation assays in *Salmonella typhimurium* (Seed, 1982; Rubin et al., 1979; Kozumbo et al., 1982). DEP was negative in mammalian cell chromosomal aberration assays (Ishidate and Odashima, 1977; Tsuchiya and Hattori, 1977). Research indicates that DEP is hydrolyzed to monoesters (Rowland et al., 1977). There is limited evidence that DEP is a weak inducer of peroxisome proliferation (U.S. EPA, 1987).

**II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)**

**II.D.1. EPA DOCUMENTATION**

U.S. EPA. 1987. Drinking Water Criteria Document for Phthalic Acid Esters. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. External Review Draft.

**II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)**

The 1987 Drinking Water Criteria Document for Phthalic Acid Esters has received OHEA review.

Agency Work Group Review: 08/26/87

Verification Date: 08/26/87

**II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)**

Welford C. Roberts / ODW -- (202)260-7589 / FTS 260-7589

Lynn Papa / ORD -- (513)569-7523 / FTS 684-7523

(REGS) Regulations:

III. HEALTH HAZARD ASSESSMENTS FOR VARIED EXPOSURE DURATIONS

IV. U.S. EPA REGULATORY ACTIONS

IV.A. CLEAN AIR ACT (CAA)

No data available

**IV.F. RESOURCE CONSERVATION AND RECOVERY ACT (RCRA)**

IV.F.1. RCRA APPENDIX IX, for Ground Water Monitoring

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

Option? CAS/100414

File: 1 Count: 1

Option? TYPE 1/2

File 1; Entry 1; Accession No. 1051

(CAS) CAS Registry Number: 100-41-4

(MAT) Material Name: Ethylbenzene

(SYN) Synonyms:

AETHYLBENZOL;  
BENZENE, ETHYL;  
EB;  
ETHYLBENZEEN;  
Ethylbenzene;  
ETHYLBENZOL;  
ETILBENZENE;  
ETYLOBENZEN;  
NCI-C56393;  
PHENYLETHANE;  
UN 1175

(UPD) Update Date: 06-01-91

(EFF) Effective Date: 07-01-91

(STAT) Status:

STATUS OF DATA FOR Ethylbenzene

File On-Line 01-31-87

Category (section) -----	Status -----	Last Revised -----
Oral RfD Assessment (I.A.)	on-line	06-01-91
Inhalation RfC Assessment (I.B.)	on-line	03-01-91
Carcinogenicity Assessment (II.)	on-line	09-07-88
Drinking Water Health Advisories (III.A.)	on-line	03-01-88
U.S. EPA Regulatory Actions (IV.)	on-line	08-01-90

## (HAZ) Chronic Health Hazards, Noncarcinogenic:

## I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

## I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

## I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
-----	-----	-----	---	-----
Liver and kidney toxicity	NOEL: 136 mg/kg/day (converted to 97.1 mg/kg/day)	1000	1	1E-1 mg/kg/day
Rat Subchronic to Chronic Oral Bioassay	LOAEL: 408 mg/kg/day (converted to 291 mg/kg/day)			
Wolf et al., 1956				
-----				

\*Conversion Factors: 5 days/7 days; thus, 136 mg/kg/day x 5 days/7 days = 97.1 mg/kg/day

## I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Wolf, M.A., V.K. Rowe, D.D. McCollister, R.L. Hollingsworth and F. Oyen. 1956. Toxicological studies of certain alkylated benzenes and benzene. Arch. Ind. Health. 14: 387-398.

The chosen study is a rat 182-day oral bioassay in which ethylbenzene was given 5 days/week at doses of 13.6, 136, 408, or 680 mg/kg/day in olive oil gavage. There were 10 albino female rats/dose group and 20 controls.

The criteria considered in judging the toxic effects on the test animals were growth, mortality, appearance and behavior, hematologic findings, terminal concentration of urea nitrogen in the blood, final average organ and body weights, histopathologic findings, and bone marrow counts. The LOAEL of 408 mg/kg/day is associated with histopathologic changes in liver and kidney.

## I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF - 1000. The uncertainty factor of 1000 reflects 10 for both intraspecies and interspecies variability to the toxicity of this chemical in lieu of specific data, and 10 for extrapolation of a subchronic effect level to its chronic equivalent.

MF - 1.

I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

None.

I.A.5. CONFIDENCE IN THE ORAL RfD

Study: Low  
Data Base: Low  
RfD: Low

Confidence in the chosen study is low because rats of only one sex were tested and the experiment was not of chronic duration. Confidence in the supporting data base is low because other oral toxicity data were not found.

Low confidence in the RfD follows.

I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

U.S. EPA. 1980. Ambient Water Quality Criteria for Ethylbenzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-048. NTIS PB 81-117590.

U.S. EPA. 1985. Drinking Water Criteria Document for Ethylbenzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. (Public review draft)

U.S. EPA. 1985. Health Effects Assessment for Ethylbenzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Emergency and Remedial Response, Washington, DC. ECAO-CIN-H008.

The 1980 Ambient Water Quality Criteria Document for Ethylbenzene received extensive Agency and public review.

The 1985 Drinking Water Criteria Document for Ethylbenzene and the 1985 Health



Effects Assessment for Ethylbenzene received extensive Agency review with the help of selected outside scientists.

Agency RfD Work Group Review: 05/20/85

Verification Date: 05/20/85

I.A.7. EPA CONTACTS (ORAL RfD)

Jeffrey C. Swartout / ORD -- (513)569-7811 / FTS 684-7811

Christopher T. DeRosa / ORD -- (513)569-7534 / FTS 684-7534

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

I.B.1. INHALATION RfC SUMMARY

Critical Effect	Exposures*	UF	MF	RfC
-----	-----	-----	---	-----
Developmental toxicity	NOAEL: 434 mg/cu.m (100 ppm)	300	1	1E+0
	NOAEL(ADJ): 434 mg/cu.m			mg/cu.m
Rat and Rabbit Developmental Inhalation Studies	NOAEL(HEC): 434 mg/cu.m			
	LOAEL: 4340 mg/cu.m (1000 ppm)			
	LOAEL(ADJ): 4340 mg/cu.m			
Andrew et al., 1981; Hardin et al., 1981	LOAEL(HEC): 4340 mg/cu.m			
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\*Conversion Factors: MW=106.18. Assuming 25C and 760 mmHg, NOAEL(mg/cu.m) =

100 ppm x MW/24.45 = 434 mg/cu.m. For developmental effects, this concentration is not adjusted; therefore, NOAEL(ADJ) = NOAEL. The NOAEL(HEC) was calculated for a gas:extrarrespiratory effect, assuming periodicity was

attained. Since b:a lambda values are unknown for the experimental animal species (a) and humans (h), a default value of 1.0 was used for this ratio.

NOAEL(HEC) = NOAEL(ADJ) x (b:a lambda(a)/lambda(h)) = 434 mg/cu.m.

I.B.2. PRINCIPAL AND SUPPORTING STUDIES (INHALATION RfC)

Andrew, F.D., R.L. Buschbom, W.C. Cannon, R.A. Miller, L.F. Montgomery, D.W.

Phelps, et al. 1981. Teratologic assessment of ethylbenzene and 2-ethoxyethanol. Battelle Pacific Northwest Laboratory, Richland, WA. PB 83-

208074., 108.

Hardin, B.D., G.P. Bond, M.R. Sikov, F.D. Andrew, R.P. Beliles and R.W. Niemeier. 1981. Testing of selected workplace chemicals for teratogenic potential. *Scand. J. Work Environ. Health.* 7(suppl 4): 66-75.

Inhalation experiments were conducted with Wistar rats (n=78-107/concentration) and New Zealand white rabbits (n=29-30/concentration) exposed 6 to 7 hours/day, 7 days/week during days 1-19 and 1-24 of gestation, respectively, to nominal concentrations of 0, 100, or 1000 ppm (434 or 4342 mg/cu.m) (Andrew et al., 1981). A separate group of rats was exposed pregestationally for 3 weeks prior to mating and exposure was continued into the gestational period. Actual concentrations were within 10% of target concentrations. All pregnant animals were sacrificed 1 day prior to term (21 days for rats; 30 days for rabbits). Maternal organs (liver, lungs, kidney, heart, spleen, adrenals, ovaries, and brain) were examined histopathologically. Uteri were examined and fetuses were weighed, sexed, and measured for crown-to-rump length, and examined for external, internal and skeletal abnormalities. For statistical analyses, the litter was chosen as the experimental unit.

Ethylbenzene did not elicit embryotoxicity, fetotoxicity, or teratogenicity in rabbits at either exposure level. There were no significant incidences of major malformations, minor anomalies, or common variants in fetal rabbits from exposed groups. Maternal toxicity in the rabbits was not evident. There was no evidence of histologic damage in any of the dams' organs. The principal observation noted by the investigators was a reduced number of live rabbit kits per litter ( $p < 0.05$ ) at both exposure levels when evaluated by ANOVA and Duncan's Multiple Range Test. The number of live kits per litter in the air-exposed controls was reported as 8 (3+/-s.d.), compared with 7 (3+/-s.d.) for each exposure group. However, if one recalculates the data presented in Table 9 of Andrew et al. (1981), the number of live kits per litter for the low concentration (100 ppm) was 8 rather than 7 as presented in the paper. Since the number of live kits per litter at the high concentration was 7, this may suggest an effect at 1000 ppm, but not at 100 ppm. However, the number of implantations per litter and the number of dead or resorbed per litter were not different from controls. Prenatal mortality ranged from 5 to

8% and preimplantation loss ranged from 18 to 27%. Neither indicated a concentration-related intrauterine mortality. The results of the rabbit study are indicative of a NOAEL of 100 ppm based on a lack of developmental effects in rabbits. The NOAEL(HEC) is 434 mg/cu.m.

In rats exposed only during gestation, there were no histopathological effects in any of the maternal organs examined. There was no effect on fertility or on any of the other measures of reproductive status. The principal observation in fetuses was an increased incidence ( $p < 0.05$ ) of supernumerary and rudimentary ribs in the high exposure group and an elevated incidence of extra ribs in both the high and 100 ppm groups. Both absolute and relative liver, kidney, and spleen weights were significantly increased in pregnant rats from the 1000 ppm group.

Groups of female rats were also exposed for 3 weeks prior to mating and exposure was continued during gestation. Like the 1000-ppm group exposed only during gestation, there was also an increased incidence of extra ribs ( $p < 0.05$ ) in the pre-gestationally exposed high exposure group. However, an increased incidence was not seen at 100 ppm in those exposed pre-gestationally, in contrast to the comparable group exposed only during gestation. There was no increase in rudimentary ribs in either of exposed groups. When extra and rudimentary ribs were grouped together, there was no significant increase in supernumerary ribs in either of the exposed groups. The apparent discrepancy in the incidence of supernumerary ribs between the pregestationally-exposed group and those exposed only during gestation may be based, in part, on the fewer numbers of litters examined in the pregestationally-exposed group. There were no effects on fertility or on any of the other measures of reproductive status. No fetal toxicity was noted at either exposure level. Body weights, placental weights, and sex ratios were within normal limits. Absolute and relative liver and spleen weights were significantly increased in pregnant rats from the 1000 ppm group; only relative kidney weight was increased significantly. There were no histopathological effects in any of the organs examined.

Skeletal variants were seen at both 434 and 4342 mg/cu.m in the rats with the effects at 432 mg/cu.m being reduced compared with those occurring at 4342 mg/cu.m. By themselves, the effects are marginally adverse, even at 4342

mg/cu.m. However, a weight-of-evidence approach, noting a cluster of other mild effects at 4342 mg/cu.m, is used to determine that 1000 ppm is a LOAEL.

The skeletal variations are considered along with evidence of slightly reduced litter size in rabbits at 4342 mg/cu.m and an increase in "% skeletal retarded fetuses" at 600 mg/cu.m (Ungvary and Tatrai, 1985). Additional support for this position is derived from the observations of somewhat elevated maternal liver, kidney, and spleen weights (Andrew et al., 1981).

#### I.B.3. UNCERTAINTY AND MODIFYING FACTORS (INHALATION RfC)

UF = 300. The uncertainty factor of 300 reflects a factor of 10 to protect unusually sensitive individuals, 3 to adjust for interspecies conversion and 10 to adjust for the absence of multigenerational reproductive and chronic studies.

MF = 1.

#### I.B.4. ADDITIONAL STUDIES / COMMENTS (INHALATION RfC)

Ungvary and Tatrai (1985) exposed CFY rats (n=17-20) to levels of 600, 1200, or 2400 mg/cu.m for 24 hours/day during days 7 to 15 of gestation. CFLP mice (n=20) were exposed to 500 mg/cu.m for 24 hours/day from gestational days 6 to 15 or for 3 days intermittently for 4 hours/day for days 6-15. It is not clear from the description if the results pertain to the continuous exposure or the intermittent exposure. New Zealand rabbits (n=3-9) were exposed for 24 hours/day to concentrations of 500 or 1000 mg/cu.m from gestational days 7 to 20. Untreated animals and those exposed to air only served as controls.

It was stated that maternal toxicity (unspecified species) was moderate and concentration-dependent; however, no data were presented to support this statement. Maternal weight gain was reported to have decreased for rabbits exposed to 1000 mg/cu.m. It was reported that rabbits exposed to 1000 mg/cu.m exhibited mild maternal toxicity manifested by reduced weight gain. However, the percent weight gain was not reported. There were no data for developmental endpoints in the 1000-ppm group because there were no live fetuses. One dam had died and three others aborted in this exposure group. Four dams had total resorptions. However, four other compounds in addition to ethyl benzene were

tested at 1000 mg/cu.m and all caused spontaneous abortions at this level.

Thus, the results are not clearly indicative of a treatment-related effect.

This observation, coupled with the lack of any indication of abortions in rabbits in the Hardin et al. (1981) study, suggests that this effect in rabbits is not treatment-related.

Ungvary and Tatnai (1985) did observe a significant reduction in the mean female fetal weight in rabbit dams exposed 24 hours/day to 500 mg/cu.m. Andrew et al. (1981) did not observe such an effect in rabbits exposed up to 4348 mg/cu.m. These conflicting results in rabbits might be attributable to differences in study design.

Postimplantation loss (% dead or resorbed fetuses), and exposure-related skeletal retardation were significantly elevated ( $p < 0.05$ ) in rats at all exposure levels with one exception. Exposure to 600 mg/cu.m for 6 hours/day (it was not stated if this was a single exposure or the exposure duration on each day of gestation) did not result in any statistically significant fetal effects although there was increased incidence of dead/resorbed fetuses, lower weight of fetuses, and skeletal retarded fetuses. In the 24-hour/day exposure groups, malformations characterized as "anomalies of the uropoietic apparatus" and an increased incidence of extra ribs were significantly increased only at the highest exposure level. No data were presented on the anomalies of the uropoietic apparatus. There was a significant ( $p < 0.05$ ) increase in skeletal retardation and fetal resorption in all continuous exposure groups although the concentration-response was shallow. The percent skeletal retarded fetuses, for example, at exposure concentrations of 600, 1200, and 2400 mg/cu.m was 26, 30, and 35%, respectively; the incidence in controls was 13%. These results in rats suggest a LOAEL(NEC) of 2400 mg/cu.m for extra ribs in the absence of demonstrable maternal toxicity.

In mice, an increased incidence of "anomalies of the uropoietic apparatus" was the only observation, but no data were presented. There was no discussion concerning maternal toxicity.

A 90-day subchronic inhalation study was conducted in F344/N rats (n=10/sex/group) and B6C3F1 mice (n=10/sex/group) that were exposed to 0, 100, 250, 500, 750, and 1000 ppm (0, 434, 1086, 2171, 3257, and 4343 mg/cu.m) 6

hours/day, 5 days/week (NTP, 1988; 1989; 1990). The duration-adjusted values were 0, 77.5, 194, 388, 582, and 776 mg/cu.m, respectively. The test atmosphere concentrations monitored by gas chromatography were within a 10% range of the target concentrations. At study termination, necropsies were conducted on the lung, liver, kidney, heart, testes, and thymus with organ weight measurements. Clinical chemistry data were obtained for rats. Histopathological examinations were conducted on all animals in the high concentration groups and in controls; animals in the lower concentration groups were evaluated when lesions were observed until no observed effects were seen. Sperm morphology and vaginal cytology tests were performed. There were no mortalities, exposure-related clinical signs of toxicity, or significant adverse effects on body weight in any of the exposed rats or mice.

In rats, hematology parameters were unaffected. Of the liver enzymes evaluated, only serum alkaline phosphatase (SAP) activity was significantly reduced in a concentration-related manner (at 500 ppm and above) for both sexes with a greater sensitivity in females. The significance of this decrease is not clear since in liver damage, SAP levels usually increase. The investigators suggested the decrease may be due to reduced water and food intake. No liver histopathology was noted for any exposure group. Significant concentration-related increases in absolute liver weights occurred in males at 250 ppm and higher (12.5, 17.3, 22.0, and 23.6% at 250, 500, 750, and 1000 ppm, respectively); in females the lowest concentration at which an increase in absolute liver weight was seen was in the 500-ppm group (11.8%). The increase in the 750- and 1000-ppm groups was 11.5 and 15.8%, respectively. Relative liver weights were significantly increased in all male exposure groups except the 100-ppm group while all female exposure groups except the two lowest groups showed significant increases. Absolute kidney weight in males significantly increased only in the 500- and 750-ppm groups; relative weight was increased in the three highest exposure groups. In females, both absolute and relative kidney weights increased significantly in the three highest exposure groups. Regeneration of renal tubules in the kidneys of male rats only was seen in all groups including controls. The severity of the lesions was greatest in the rats at in the high-exposure group.

The most significant gross observation in rats was the presence of enlarged bronchial and/or mediastinal lymph nodes, but these observations were not dose-related. The incidence for minimal lung inflammation in male rats

was 0/10, 3/10, 9/10, 9/10, 8/10, and 10/10 for the 0-, 100-, 250-, 500-,

750-, and 1000-ppm exposure groups, respectively. Microscopically, this enlargement was attributable to an increase in normal constituents of the lymph nodes characterized by accumulations of macrophages, lymphocytes, neutrophils, and plasma cells. It was the opinion of the NTP Pathology Working Group (PWG) that hyperplasia of the lymph nodes and lower respiratory

tract was typical of an infectious agent with an associated active immune response rather than ethylbenzene exposure (NTP, 1989). This diagnosis was

supported by the following observations: an uneven distribution of lesions

among and within groups; foci of airway inflammation were randomly distributed throughout the lungs; considerable variability in severity within groups; and

there was no consistent concentration-response relationship. No lesions were seen in the nasal cavity. PWG described these lesions as not typical of

the type of lesions which occurs with known pulmonary irritants. These lesions were not found in control animals, which were housed in separate rooms. No infectious agent was identified upon serologic examination. In the draft NTP technical report (NTP, 1990), the inflammatory lung lesions were

described as probably unrelated to exposure. Antibodies to common rodent respiratory tract viruses were not detected. However, only sera from control

rats were sampled. Lesions morphologically indistinguishable from those in

this study have been seen in control and treatment groups of rats from other

inhalation and dosed feed studies (NTP, 1990). The PWG recommended that this

effect be reevaluated in another study.

In mice, no significant exposure-related gross or histopathological observations were noted at terminal necropsy of any organs, including the lung. The only exposure-related effects were significantly elevated absolute

and relative liver weight in both sexes of mice at of 750 and 1000 ppm and

significantly elevated relative kidney weight of the females exposed to 1000

ppm. There were no significant histopathological changes or function test

alterations in either liver or kidney of either sex.

The NTP peer review of the subchronic study took place on November 20,

1990 at Research Triangle Park. The NTP Board of Scientific Counselors' panel of experts agreed with the conclusions of the NTP report that there were no

indications of toxicity due to ethyl benzene. A 2-year lifetime study in both

rats and mice has been initiated and exposures have been conducted through 7 months. No serial sacrifices are planned and results are not expected prior to 1992.

Clark (1983) exposed Wistar rats (n=18/sex/group) (12-13 weeks old) to 0 and 100 ppm (0 and 434 mg/cu.m) reagent grade ethylbenzene 6 hours/day, 5 days/week for 12 weeks. The duration-adjusted values were 0 and 77.5 mg/cu.m. Clinical observations, body weight, food intake, hematology, urinalysis, organ weights, and histopathology of all major organs (including the lung and nasal cavity) were used as parameters to assess toxicity. No statistically significant effects were observed at 100 ppm. There were no differences from controls in the liver enzymes, including SAP. While slight bile duct hyperplasia was seen in 15/18 exposed males and 14/18 exposed females, hyperplasia was also common in controls (10/18 females and 8/18 males), and these observations were not statistically significant. The results of this study suggest a NOAEL of 100 ppm. The NOAEL(HEC) is 77.5 mg/cu.m. The results are in general agreement with the findings of the NTP study in F344 rats.

Wolf et al. (1956) exposed rats (n=10-25/sex/group) to 400, 600 or 1250 ppm (1737, 2606, or 5428 mg/cu.m) ethylbenzene 7 hours/day, 5 days/week for about 6 months. The duration-adjusted values were 0, 362, 542, and 1131 mg/cu.m, respectively, using the 7-hour duration. Exposure ranged from 186 to 214 days. Male rats only were also exposed to 2200 ppm (9554 mg/cu.m) for 7 hours/day, 5 days/week for about 5 months. The duration-adjusted value was 1990 mg/cu.m. Histopathology was performed on a variety of organs including the lung. Data on liver and kidney weights and histopathology were not presented; these parameters were discussed only in descriptive terms. Repeated exposure of rats, guinea pigs, and rhesus monkeys was examined.

Growth was depressed moderately in male rats at 2200 ppm. Liver and kidney weights in rats were increased slightly in all exposed groups compared with matched controls, and rats exposed to 1250 and 2200 ppm developed histopathological changes manifested as cloudy swelling of the liver and renal tubules and testicular degeneration. The data indicate a NOAEL for liver histopathology at 600 ppm (542 mg/cu.m). However, no incidence data was reported. Since it is not clear that these effects are adverse when taken in context with the results of the NTP study, a NOAEL or LOAEL is not identified.



Guinea pigs (5-10/sex/group) and rabbits (1-2/sex/group) were exposed to 0, 400, or 600 ppm (duration-adjusted concentrations of 0, 362, or 542 mg/cu.m, respectively) ethylbenzene 7 hours/day, 5 days/week for about 6 months. Only females were exposed to 1250 ppm (duration-adjusted value of 1131 mg/cu.m). Growth was depressed in female guinea pigs exposed to 1250 ppm. Liver weight was described as slightly increased only in the 600-ppm exposure group. The study does not clearly indicate 600 ppm as a LOAEL so the NOAEL for guinea pigs is designated at 600 ppm. The NOAEL(HEC) is 542 mg/cu.m. Other than an observation of slight degeneration of the testicular germinal epithelium in the male rabbit at 600 ppm, there were no adverse effects reported for rabbits of either sex.

One male Rhesus monkey was exposed to 600 ppm (duration-adjusted value of 542 mg/cu.m) and two females were exposed to 400 ppm (duration-adjusted value of 362 mg/cu.m). A slight degeneration of the testicular germinal epithelium and increased liver weight was observed in the male monkey. No effects were reported for the female rhesus monkeys.

The small number of rabbits and monkeys preclude identification of NOAEL and LOAEL values for these species.

Cragg et al. (1989) exposed B6C3F1 mice (n=5/sex/group) and F344 rats (n=5/sex/group) to actual concentrations of 0, 99, 382, and 782 ppm (0, 430, 1659, and 3396 mg/cu.m) 6 hours/day, 5 days/week for 4 weeks. The duration-adjusted values were 0, 77, 296, 606 mg/cu.m, respectively. In the same study, New Zealand White rabbits (n=5/sex/group) were exposed to actual concentrations of 0, 382, 782, or 1610 ppm (0, 1659, 3396, or 6992 mg/cu.m). The duration-adjusted values were 0, 296, 606 and 1249 mg/cu.m, respectively.

No changes were evident in mortality, clinical chemistry parameters, urinalysis, nor were there treatment-related gross or histopathological findings. Urinalysis was not performed on rabbits and clinical chemistry parameters were not performed on mice. Liver enzymes measured included AP.

Hematology was performed on all species. Histopathology was only conducted on the high concentration animals except all rabbits' testes were examined. There was no liver histopathology in any of the species.

In the 382-ppm exposure group, rats exhibited sporadic incidences of salivation and lacrimation. (These observations were not noted in the NTP

subchronic study). Absolute liver weights were significantly increased in male rats; relative weight was increased at 782 ppm. In females, absolute liver weight was significantly increased at 782 ppm and relative weight at both concentrations. Male rats of the 782 ppm group had a significant ( $p < 0.05$ ) increase in platelets while females only had a significant ( $p < 0.05$ ) increase in total leukocytes.

In mice, females showed a statistically significant increase in absolute, but not relative liver weight, at 782 ppm. There were no significant liver weight changes in male mice. Both males and females exhibited an increase in liver weight relative to brain weight at 782 ppm only. Rabbits showed no changes in liver weight ratios at any exposure level.

Since there were no adverse histopathological findings for the liver, a NOAEL of 782 ppm is identified for rats and mice. The NOAEL(HEC) is 606 mg/cu.m. The NOAEL for rabbits is 1610 ppm; the NOAEL(HEC) is 1249 mg/cu.m.

Elovaara et al. (1985) found concentration-related increases in drug-metabolizing enzymes of liver and kidney, with corresponding ultrastructural alterations in a subchronic inhalation study with rats. Male Wistar rats ( $n=5$ /group) were exposed to 0, 50, 300, or 600 ppm (0, 217, 1302, or 2604 mg/cu.m) ethylbenzene 6 hours/day, 5 days/week for 2, 5, 9, or 16 weeks. The duration-adjusted values were 0, 38.7, 233, and 465 mg/cu.m, respectively. The liver was the only organ examined histologically (light and electron microscopy). There were no changes in liver weight at any concentration. After 16 weeks exposure, NADPH-cytochrome reductase and UDPG-transferase were significantly elevated at 300 and 600 ppm. Aminopyrine N-demethylase and 7-ethoxycoumarin-O-deethylase (7-ECDE) were elevated at all exposure levels. The elevation in UDPG-transferase was exposure-related and may signify glucuronidation of ethylbenzene metabolites during detoxication. Electron microscopy also showed changes in hepatocyte ultrastructure [e.g., smooth endoplasmic reticulum (SER) proliferation, slight degranulation of rough endoplasmic reticulum] at all exposure levels beginning 2 to 9 weeks after exposure. Necrosis was not observed nor were there any increases in serum alanine aminotransferase. SAP was not measured. The proliferation of SER is

consistent with enzyme induction. At 16 weeks, changes in ultrastructure were mainly confined to the high-exposure group. There was no effect of exposure on hepatic glutathione (GSH) content. Significant increases in relative kidney weight only were reported following 2 and 9, but not at 16 weeks of exposure to 600 ppm. Kidney 7-ECDE, and UDPG transferase activities showed statistically significant and exposure-related increases at all exposure levels.

In the absence of histologic evidence of damage, changes in absolute or relative liver weight, and no effect on serum ALT, the microsomal enzyme induction and ultrastructural changes are considered to be adaptation phenomena. The results of this study suggest a NOAEL of 600 ppm. The NOAEL(HEC) is 465 mg/cu.m for liver and kidney. The absence of liver weight changes is not consistent with the findings of the NTP (1988) subchronic study.

Angerer and Wulf (1985) evaluated 35 workers who chronically (2-24 years, average 8.2 years) sprayed varnishes containing alkyd-phenol and polyester resins dissolved in solvent mixtures consisting principally of xylene isomers and ethylbenzene. Some of the varnishes contained lead-based pigments. The air samples from personal monitors indicated average levels of 4.0 ppm for ethylbenzene. Although workers had significantly elevated lymphocytes in addition to significantly decreased erythrocyte counts and hemoglobin levels compared with controls, these effects cannot be attributed to ethylbenzene since other compounds (e.g., xylene, methylchloroform, n-butanol, toluene, C9 hydrocarbons) were detected in some of the six workplaces evaluated.

Bardodej and Cirek (1988) carried out biomonitoring of 200 ethylbenzene production workers occupationally exposed for a mean duration of 12.2 years to unspecified concentrations of ethylbenzene and benzene over a 20-year period. The workers were evaluated twice a year and ethylbenzene metabolites measured. No statistically significant differences in hematological effects (e.g., RBC, WBC, leukocyte and platelet counts) or liver function tests (e.g., aminotransferase and/or SAP and LDH activities and bilirubin tests) were observed between exposed and nonexposed workers.

#### I.B.5. CONFIDENCE IN THE INHALATION RfC

Study: Low  
Data Base: Low  
RfC: Low

The developmental study by Hardin et al. (1981) was well-conducted and indicated no clearly adverse effects in any species. The study is given a low confidence rating because higher exposure levels may have provided more information on the potential for maternal toxicity and developmental effects.

The data base is given a low rating since although other studies have examined a variety of other endpoints (e.g., liver and lung), by histopathology in rats and mice, there are no chronic studies and no multi-generation developmental studies. These latter studies would be useful to determine more conclusively the potential of ethylbenzene to affect development.

NTP does not consider observations of lung lesions in rats exposed in the NTP subchronic study to be treatment-related. However, no infectious agent has been detected. Therefore, there remains a possibility that ethylbenzene may play a role in producing lung lesions. It is anticipated that this issue will be clarified upon completion of the chronic study in progress.

In view of the previous considerations, the RfC is given a low confidence rating.

#### I.B.6. EPA DOCUMENTATION AND REVIEW OF THE INHALATION RfC

Source Document -- This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation -- U.S. EPA, 1984; 1985; 1987.

Agency Work Group Review : 09/19/90, 12/20/90

Verification Date: 12/20/90

#### I.B.7. EPA CONTACTS (INHALATION RfC)

Mark Greenberg / ORD -- (919)541-4156 / FTS 629-4156

Annie M. Jarabek / ORD -- (919)541-4847 / FTS 629-4847

(CAR) Carcinogenicity Assessment:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity.

Basis -- nonclassifiable due to lack of animal bioassays and human studies.

II.A.2. HUMAN CARCINOGENICITY DATA

None.

II.A.3. ANIMAL CARCINOGENICITY DATA

None. NTP has plans to initiate bioassay. Metabolism and excretion studies at 3.5, 35 and 350 mg/kg are to be conducted as well.

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

The metabolic pathways for humans and rodents are different (Engstrom et al., 1984). Major metabolites in humans, mandelic acid and phenylglyoxylic acid, are minor metabolites in rats and rabbits (Kiese and Lenk, 1974). The major animal metabolites were not detected in the urine of exposed workers (Engstrom et al., 1984).

Ethylbenzene at 0.4 mg/plate was not mutagenic for Salmonella strains TA98, TA1535, TA1537 and TA1538 with or without Aroclor 1254 induced rat liver homogenates (S9) (Nestmann et al., 1980). Ethylbenzene was shown to increase the mean number of sister chromatid exchanges in human whole blood lymphocyte culture at the highest dose examined without any metabolic activation system (Norppa and Vainio, 1983).

Dean et al. (1985) used a battery of short-term tests including bacterial mutation assays, mitotic gene conversion in *Saccharomyces cerevisiae* JD1 in the presence and absence of S9 and chromosomal damage in a cultured rat liver cell line. Ethylbenzene was not mutagenic in the range of concentrations tested (0.2, 2, 20, 50 and 200 ug/plate) for *S. typhimurium* TA98, TA100, TA1535, TA1537 and TA1538 or for *Escherichia coli* WP2 and WP2uvrA. Ethylbenzene also showed no response in the *S. cerevisiae* JD1 gene conversion assay. In contrast, ethylbenzene hydroperoxide showed positive responses with *E. coli* WP2 at 200 ug/plate in the presence of S9 and an equally

significant response with the gene conversion system of yeast.

## II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1980. Ambient Water Quality Criteria Document for Ethylbenzene.

Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-048. NTIS PB 81-117590.

U.S. EPA. 1984. Health Effects Assessment for Ethylbenzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Emergency and Remedial Response, Washington, DC. EPA/540/1-86/008.

U.S. EPA. 1987. Drinking Water Criteria Document for Ethylbenzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.

### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The Ambient Water Quality Criteria Document and the Health Assessment Document have received Agency and external review. The Drinking Water Criteria Document has been extensively reviewed.

Agency Work Group Review: 10/07/87

Verification Date: 10/07/87

### II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Arthur S. Chiu / ORD -- (202)475-6764 / FTS 475-6764

Annette Gatchett / ORD -- (513)569-7813 / FTS 684-7813

## (HA) Hazard Assessment:

### III. HEALTH HAZARD ASSESSMENTS FOR VARIED EXPOSURE DURATIONS

#### III.A. DRINKING WATER HEALTH ADVISORIES

##### III.A.1. ONE-DAY HEALTH ADVISORY FOR A CHILD

One-day HA --  $3.2E+1$  mg/L

NOAEL -- 31.8 mg/kg/day

UF -- 10 (allows for intrahuman variability with the use of a NOAEL from a human study)

Assumptions -- 1 L/day water consumption for a 10-kg child

Principal Study -- Bardodej and Bardodejova, 1970

No adverse health effects were observed in human volunteers exposed to ethylbenzene by inhalation at a concentration of 100 ppm (435 mg/cu.m) for 8 hours. Based on the conditions of exposure and an assumed absorption factor of 64%, this is equivalent to a NOAEL of 31.8 mg/kg/day.

#### III.A.2. TEN-DAY HEALTH ADVISORY FOR A CHILD

Appropriate data for calculating a Ten-day HA are not available. Therefore, the Ten-day HA has been calculated from the One-day HA by dividing the One-day HA of 32 mg/L by 10. The Ten-day HA is therefore 3.2 mg/L.

#### III.A.3. LONGER-TERM HEALTH ADVISORY FOR A CHILD

Appropriate data for calculating a Longer-term HA are not available. It is recommended that the modified DWEL (adjusted for a 10-kg child) of 0.97 mg/L (rounded to 1 mg/L) be used as the Longer-term HA.

#### III.A.4. LONGER-TERM HEALTH ADVISORY FOR AN ADULT

Appropriate data for calculating a Longer-term HA are not available. It is recommended that the DWEL of 3.4 mg/L be used as the Longer-term HA for the 70-kg adult.

#### III.A.5. DRINKING WATER EQUIVALENT LEVEL / LIFETIME HEALTH ADVISORY

DWEL --  $3.4E+0$  mg/L

Assumptions -- 2 L/day water consumption for a 70-kg adult

RfD Verification Date - 05/20/85 (see Section I.A. of this file)

Lifetime HA --  $6.8E-1$  mg/L

Assumptions -- 20% exposure by drinking water

Principal Study -- Wolf et al., 1956 (This study was used in the derivation

of the chronic oral RfD; see Section I.A.2.)

### III.A.6. ORGANOLEPTIC PROPERTIES

Taste perception threshold (water) -- 0.029 mg/L.

Odor perception threshold (water) -- 0.029 mg/L.

Odor perception threshold (air) -- 0.062 mg/L.

### III.A.7. ANALYTICAL METHODS FOR DETECTION IN DRINKING WATER

Analysis of ethylbenzene is by a purge-and-trap gas chromatographic procedure used for the detection of volatile organic compounds in water. Confirmatory analysis is by mass spectrometry.

### III.A.8. WATER TREATMENT

Ethylbenzene is most effectively removed from water by air stripping. Adsorption on activated carbon is at least partially effective in the removal of ethylbenzene from solution. Conventional treatment processes may also be effective.

### III.A.9. DOCUMENTATION AND REVIEW OF HAS

U.S. EPA. 1985. Final Draft of the Drinking Water Criteria Document on Ethylbenzene. Office of Drinking Water, Washington, DC.

EPA review of HAS in 1985.

Public review of HAS following notification of availability in October, 1985.

Scientific Advisory Panel review of HAS in January, 1986.

Preparation date of this IRIS summary -- 06/24/87

### III.A.10. EPA CONTACTS

Charles O. Abernathy / ODW -- (202)382-5374 / FTS 382-5374

Edward V. Ohanian / ODW -- (202)382-7571 / FTS 382-7571

(REGS) Regulations:

## IV. U.S. EPA REGULATORY ACTIONS

### IV.B. SAFE DRINKING WATER ACT (SDWA)

#### IV.B.1. MAXIMUM CONTAMINANT LEVEL GOAL (MCLG) for Drinking Water



Value (status) -- 0.68 mg/L (Proposed, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- An MCLG of 0.68 mg/L for ethylbenzene is proposed based upon a provisional DWEL of 3.4 mg/L and an assumed drinking water contribution of 20%. A DWEL of 3.4 mg/L was calculated from a NOAEL of 136 mg/kg/day for histopathological changes (not specified) in rats (6-month oral study) with an uncertainty factor of 1000, conversion factor of 5/7 and consumption of 2 L of water/day.

Reference -- 50 FR 46936 Part IV (11/13/85)

EPA Contact -- Charles Abernathy / Criteria and Standards Division, ODW / (202)382-7571 / FTS 382-7571; or Drinking Water Hotline / (800)426-4791

#### IV.C. CLEAN WATER ACT (CWA)

##### IV.C.1. AMBIENT WATER QUALITY CRITERIA, Human Health

Water and Fish Consumption: 1.4 mg/L

Fish Consumption Only: 3.28 mg/L

Considers technological or economic feasibility? -- NO

Discussion -- The WQC of 1.4 mg/L is based on consumption of contaminated aquatic organisms and water. A WQC of 3.28 mg/L has also been established based on consumption of contaminated aquatic organisms alone.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS / (202)475-7315 / FTS 475-7315

##### IV.C.2. AMBIENT WATER QUALITY CRITERIA, Aquatic Organisms

#### Freshwater:

Acute -- 32,000 ug/L (LEL)

Chronic -- None

#### Marine:

Acute -- 430 ug/L (LEL)

Chronic -- None

Considers technological or economic feasibility? -- NO

Discussion -- Water quality criteria for the protection of aquatic life are derived from a minimum data base of acute and chronic tests on a variety of aquatic organisms. The "(LEL)" after the value indicates that the minimum data were not available and the concentration given is not a criteria value but the lowest effect level found in the literature.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS /  
(202)475-7315 / FTS 475-7315

#### IV.F. RESOURCE CONSERVATION AND RECOVERY ACT (RCRA)

##### IV.F.1. RCRA APPENDIX IX, for Ground Water Monitoring

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

#### IV.G. SUPERFUND (CERCLA)

##### IV.G.1. REPORTABLE QUANTITY (RQ) for Release into the Environment

Value (status) -- 1000 pounds (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ is based on aquatic toxicity, as established under Section 311(b)(4) of the Clean Water Act (40 CFR 117.3), and ignitability.

Available data indicate that the aquatic 96-Hour Median Threshold Limit for ethylbenzene is between 10 and 100 ppm. The closed-cup flash point is less than 100F and the boiling point is >100F.

Reference -- 50 FR 13456 (04/04/85)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

Option? TYPE 10/2

File 10; Entry 1; Accession No. 1444

(CAS) CAS Registry Number: 206-44-0

(MAT) Material Name: Fluoranthene

(SYN) Synonyms:

1,2-BENZACENAPHTHENE;  
BENZENE, 1,2-(1,8-NAPHTHALENEDIYL)-;  
BENZENE, 1,2-(1,8-NAPHTHYLENE)-;  
BENZO(JK) FLUORENE;  
FLUORANTHENE;  
HSDB 5486;  
IDRYL;  
1,2-(1,8-NAPHTHYLENE) BENZENE;  
NSC 6803;  
RCRA WASTE NUMBER U120

(UPD) Update Date: 07-01-91

(EFF) Effective Date: 07-01-91

(STAT) Status:

STATUS OF DATA FOR Fluoranthene

File On-Line 09-01-90

Category (section)	Status	Last
Revised		
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Oral RfD Assessment (I.A.)	on-line	
07-01-91		
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	
12-01-90		
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	no data	
Supplementary Data (V.)	no data	

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(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.A.1. ORAL RfD SUMMARY

Critical Effect RfD	Experimental Doses*	UF	MF
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Nephropathy, increased 4E-2 liver weights, hema- mg/kg/day tological alterations, and clinical effects	NOAEL: 125 mg/kg/day  LOAEL: 250 mg/kg/day	3000	1

Mouse Subchronic Study

U.S. EPA, 1988

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\*Conversion Factors: None

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

U.S. EPA. 1988. 13-Week mouse oral subchronic toxicity study.  
Prepared by  
Toxicity Research Laboratories, Ltd., Muskegon, MI for the  
Office of Solid  
Waste, Washington, DC.

Male and female CD-1 mice (20/sex/group) were gavaged for 13 weeks with 0,

125, 250, or 500 mg/kg/day fluoranthene. A fifth group of mice (30/sex) was established in the study for baseline blood evaluations. Body weight, food consumption, and hematological and serum parameter values were recorded at regular intervals during the experiment. At the end of 13 weeks, the animals were sacrificed and autopsied, which included organ weight measurement and histological evaluation. All treated mice exhibited nephropathy, increased

salivation, and increased liver enzyme levels in a dose-dependent manner.

However, these effects were either not significant, not dose-related, or not

considered adverse at 125 mg/kg/day. Mice exposed to 500 mg/kg/day had

increased food consumption and increased body weight. Mice exposed to 250 and

500 mg/kg/day had statistically increased SGPT values and increased absolute

and relative liver weights. Compound-related microscopic liver lesions

(indicated by pigmentation) were observed in 65 and 87.5% of the mid- and

high-dose mice, respectively. Based on increased SGPT levels, kidney and

liver pathology, and clinical and hematological changes, the LOAEL is

considered to be 250 mg/kg/day, and the NOAEL is 125 mg/kg/day.

#### I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF = 3000. An uncertainty factor of 3000 reflects 10 for interspecies

conversion, 10 for intraspecies variability, and 30 for use of a subchronic

study for chronic RfD derivation, and for lack of supporting reproductive/developmental toxicity data and toxicity data in a second species.

MF = 1.

#### I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

A developmental study was performed in which fluoranthene was administered

once via intraperitoneal injection to pregnant C57/B6 mice on gestational day

6, 7, 8 or 9 (Irvin and Martin, 1987). An increased rate of embryo resorption

was observed. The data were reported in an abstract, but a complete report

was not located. No inhalation studies were located.

IARC (1983) cites several acute studies in which fluoranthene was

administered to mice or rats intraperitoneally. No adverse effects were

observed; however, only survival or body weight was monitored. Gerarde (1960,

cited by IARC, 1983) administered 500 mg/kg/day for 7 days to mice, and Haddow

et al. (1937) administered a single 30 mg dose of fluoranthene to rats.

#### I.A.5. CONFIDENCE IN THE ORAL RfD

Study: Medium  
Data Base: Low  
RfD: Low

Confidence in the principal study is medium, as it is a well-designed study that identified both a LOAEL and a NOAEL for several sensitive endpoints using an adequate number of animals. Confidence in the data base is low; developmental, reproductive, or toxicity data in a second species following oral exposure to fluoranthene has not been adequately tested. Reflecting

medium confidence in the principal study and low confidence in the database, confidence in the RfD is low.

#### I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- This assessment is not presented in any existing U.S. EPA document.

Agency Work Group Review: 01/22/86, 10/19/89, 11/15/89

Verification Date: 11/15/89

#### I.A.7. EPA CONTACTS (ORAL RfD)

Harlal Choudhury / ORD -- (513)569-7536 / FTS 684-7536

Kenneth A. Poirier / ORD -- (513)569-7553 / FTS 684-7553

(CAR) Carcinogenicity Assessment:

### II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

#### II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

##### II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity

Basis -- Based on no human data and inadequate data from animal bioassays.

## II.A.2. HUMAN CARCINOGENICITY DATA

None.

## II.A.3. ANIMAL CARCINOGENICITY DATA

Inadequate. Data from fluoranthene skin-painting bioassays was judged inadequate because no increases in tumor incidences were observed and the group sizes tested were small.

Fluoranthene has been tested as a complete carcinogen in mouse skin-painting assays at doses ranging approximately from 1.5 mg/mouse/week for 52 weeks to 100 mg/mouse/week for 82 weeks; the results of these studies have been consistently non-positive (Suntzeff et al., 1957; Wynder and Hoffmann, 1959; Hoffmann et al., 1972; Horton and Christian, 1974).

Suntzeff et al. (1957) administered a 10% solution of fluoranthene in acetone by topical application 3 times/week to unspecified numbers of CAF, Jackson, Swiss and Millerton mice. No tumors were found by 13 months. Wynder and Hoffmann (1959) administered a 0.1% solution of fluoranthene in acetone onto the backs of 20 female Swiss (Millerton) mice 3 times/week for life. No tumors were found. Hoffmann et al. (1972) administered 50 uL of a 1% fluoranthene solution to the backs of 20 female Swiss-albino Ha/ICR/Mill mice 3 times/week for 12 months. All treated mice survived and no tumors were observed. As part of the same study, 30 mice received 0.1 mg fluoranthene in 50 uL acetone every second day for a total of 10 doses. Promotion by dermal application of 2.5% croton oil in acetone was initiated 10 days later and continued for 20 weeks. A single papilloma was noted in 29 surviving mice. Horton and Christian (1974) administered 50 mg fluoranthene in decalin or in decalin:n-dodecane (50:50) to the backs of 15 male C3H mice. The mice were treated 2 times/week for 82 weeks. No skin tumors were observed.

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

In a short-term in vivo lung tumor assay by Busby et al. (1984), CD-1 mice (20-30/sex/dose) received intraperitoneal injections of dimethyl sulfoxide (DMSO) or fluoranthene in DMSO on days 1, 8, and 15 after birth; total doses were 0, 700 ug (163 mg/kg) or 3500 ug (815 mg/kg) fluoranthene. Animals were necropsied at 24 weeks of age. Visible lung tumors were tabulated at necropsy and examined histologically; all tissue masses and organs exhibiting abnormal growth were examined histologically. A statistically significant increase in the incidence of combined lung adenomas and adenocarcinomas occurred in the male-female combined high-dose group (28/48) when compared with vehicle controls (5/55). In the combined high-dose groups 80% of the lung tumors were adenomas and 20% adenocarcinomas; no adenocarcinomas occurred in the control groups. Lung tumor response in the combined low-dose groups (10/51) was not statistically different from controls. Lung tumor incidence was significantly elevated in high-dose males (20/27 vs. 1/27 controls) but not in low-dose males (7/31) or in high- or low-dose females (8/21 and 3/20, respectively, vs. 4/28 in the controls).

Fluoranthene produced positive results in mouse co-carcinogen skin-painting assays with benzo[a]pyrene. This combination of chemicals increased the formation of benzo[a]pyrene-DNA adducts (Van Duuren and Goldschmidt, 1976; Rice et al., 1988).

Barry et al. (1935) administered 300 mg fluoranthene in benzene by dermal application (number of applications not stated) to 20 mice (type unspecified). The survival rate was 35% after 6 months and 20% at 1 year. No tumors were found by 501 days. Shear (1938) administered four doses of 10 mg fluoranthene in glycerol by subcutaneous injection to strain A mice. Six out of 14 mice survived for 18 months; no tumors were found by 19 months. In a skin-painting



assay fluoranthene (100 ug) was administered to 20 Swiss albino Ha/ICR mice, 3 times/week for 1 year; 3.3% of the mice in both this group and in a similar acetone-control group tumors were observed in 3.3% of the mice in both the treated and acetone-control groups (LaVoie et al., 1979).

Evidence for mutagenicity of fluoranthene is equivocal. The results of mutagenicity assays of fluoranthene in several strains of *Salmonella typhimurium* have been positive (Kaden et al., 1979; Kinae et al., 1981; LaVoie et al., 1982; Babson et al., 1986; Bos et al., 1988) and not positive (Tokiwa et al., 1977; Kinae et al., 1981; Bos et al., 1987). Evidence for mutagenicity in mammalian cells is also equivocal: results of tests for chromosomal effects in Chinese hamster cells have been both positive (Palitti et al., 1986) and not positive (DeSaliva et al., 1988). A test for gene mutations in human lymphoblast cells was not positive (Crespi and Thilly, 1984), whereas results of tests in different mutant Chinese hamster ovary cell lines have been both positive (Hoy et al., 1984; Li, 1984) and not positive (Hoy et al., 1984).

## II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

None.

## II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

None.

## II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1990. Drinking Water Criteria Document for

Polycyclic Aromatic  
Hydrocarbons (PAHs). Prepared by the Office of Health and  
Environmental  
Assessment, Environmental Criteria and Assessment Office,  
Cincinnati, OH for  
the Office of Drinking Water, Washington, DC. Final Draft.  
ECAO-CIN-D010,  
September, 1990.

#### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The 1990 Drinking Water Criteria Document for Polycyclic  
Aromatic  
Hydrocarbons has received Agency and external review.

Agency Work Group Review: 05/03/90

Verification Date: 05/03/90

#### II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

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File 5; Entry 1; Accession No. 1435

(CAS) CAS Registry Number: 86-73-7

(MAT) Material Name: Fluorene

(SYN) Synonyms:

9H-Fluorene;  
Diphenylenemethane;  
Fluorene;  
HSDB 2165;  
Methane, diphenylene-;  
NSC 6787;  
o-BIPHENYLENEMETHANE;  
2,2'-METHYLENEBIPHENYL;  
9H-fluorene

(UPD) Update Date: 12-01-90

(EFF) Effective Date: 10-01-91

(STAT) Status:

STATUS OF DATA FOR Fluorene

File On-Line 11-01-90

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	11-01-90
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	12-01-90
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	no data	
Supplementary Data (V.)	no data	

(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
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Decreased RBC,  
packed cell volume  
and hemoglobin

NOAEL: 125 mg/kg/day  
LOAEL: 250 mg/kg/day

3000

1

4E-2  
mg/kg/day

Mouse Subchronic Study

U.S. EPA, 1989

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\*Conversion Factors: None

#### I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

U.S. EPA. 1989. Mouse oral subchronic toxicity study. Prepared by Toxicity Research Laboratories, LTD., Muskegon, MI for the Office of Solid Waste, Washington, DC.

CD-1 mice (25/sex/group) were exposed to 0, 125, 250, or 500 mg/kg/day fluorene suspended in corn oil by gavage for 13 weeks. Parameters used to assess toxicity included food intake, body weight, clinical observations, hematology and serum chemistry and gross and histopathological examinations. Increased salivation, hypoactivity, and urine-wet abdomens in males were observed in all treated animals. The percentage of mice exhibiting hypoactivity was dose-related. In mice exposed at 500 mg/kg/day, labored respiration, ptosis (drooping eyelids), and unkempt appearance were also observed. A significant decrease in red blood cell count and packed cell volume were observed in females treated with 250 mg/kg/day fluorene and in males and females treated with 500 mg/kg/day. Decreased hemoglobin concentration and increased total serum bilirubin levels were also observed in the 500 mg/kg/day group. Decreases in erythrocyte count, packed cell volume, and hemoglobin concentration were all observed at 125 mg/kg; however, these effects, although apparently dose-dependent, were not statistically significant. A significant decreasing trend in BUN and a significant increasing trend in total serum bilirubin were observed in both high-dose males and females. A dose-related increase in relative liver weight was observed in treated mice; a significant increase in absolute liver weight was also observed in the mice treated with 250 and 500 mg/kg/day fluorene. A significant increase in absolute and relative spleen and kidney weight was observed in males and females exposed to 500 mg/kg/day and males at 250 mg/kg/day. Increases in the absolute and relative liver and spleen weights in the high-dose males and females were accompanied by histopathological increases in the amounts of hemosiderin in the spleen and in the Kupffer cells of the liver. No other histopathological lesions were observed. The LOAEL is 250 mg/kg/day based on hematological effects; the NOAEL is 125 mg/kg/day.

#### I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF = 3000. An uncertainty factor of 3000 was used: 10 for use of a subchronic study for chronic RfD derivation, 10 each for inter- and intraspecies variability, and 3 for lack of adequate toxicity data in a second species and reproductive/developmental data.

MF = 1.

#### I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

Morris et al. (1960) fed 18 female Buffalo strain rats 12.3 mg fluorene/kg/day for 6 months or 13.1 mg fluorene/kg/day for 18 months. The diet in the 6-month study was composed of purified materials, low in protein and fat, and prepared in 3% propylene glycol. The diet in the longer study was composed of a mixture of natural foodstuffs in 3% corn oil. In the 6-month study, of 11 animals examined, the incidences of non-neoplastic reactions were reported by organ as follows: forestomach (acanthosis, hyperkeratosis), 5 animals; kidney (squamous metaplasia of pelvis), 7 animals; uterus (squamous metaplasia), 1 animal; small intestine (epithelial ulcer, acute), 1 animal; and liver (cirrhosis), 3 animals.

In the longer study using 18 rats, none of the effects seen in the 6-month study were observed. The only effect reported in this experiment was hyperplasia of the pituitary (predominantly chromophobe cells) in two animals.

It appears that the effects observed in the 6-month study were related to either dietary composition or propylene glycol, since none of these effects were observed after 18 months at a similar dosage using a different diet and vehicle. Consequently, this study is not considered acceptable as a basis for chronic RfD derivation.

No other studies on the toxicity of orally administered fluorene were located.

#### I.A.5. CONFIDENCE IN THE ORAL RfD

Study: Medium  
Data Base: Low  
RfD: Low

Confidence in the principal study is medium: it is a well-designed study that examined and identified both a LOAEL and NOAEL for several sensitive endpoints using an adequate number of animals. Confidence in the data base is low; developmental, reproductive, and chronic toxicity following oral exposure to fluorene have not been tested, and a NOAEL was not identified. Confidence in the RfD is accordingly low.

#### I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation - U.S. EPA, 1987

Agency Work Group Review: 10/19/89, 11/15/89

Verification Date: 11/15/89

#### I.A.7. EPA CONTACTS (ORAL RfD)

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(CAR) Carcinogenicity Assessment:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity

Basis -- Based on no human data and inadequate data from animal bioassays.

II.A.2. HUMAN CARCINOGENICITY DATA

None.

II.A.3. ANIMAL CARCINOGENICITY DATA

Inadequate. Morris et al. (1960) fed female buffalo rats a diet containing 0.05% fluorene in 3% corn oil for approximately 18 months or in propylene glycol for about 6 months (approximately 11 mg/kg/day). Various types of tumors occurred in controls and exposed animals at approximately the same incidences, ranging from 6 to 34%. No statistical analysis was reported.

Studies of fluorene for complete carcinogenic activity, initiating activity or co-carcinogenicity with 3-methylcholanthrene in mouse skin painting assays were not positive or were inconclusive (Kennaway, 1924; Riegel et al., 1951; LaVoie et al., 1979, 1981).

No injection site tumors occurred within 18 months in 10 strain A mice after seven subcutaneous injections of 10 mg fluorene in glycol (Shear, 1938). No control groups appear to have been utilized in this study.

Wilson et al. (1947) fed two groups of albino rats various concentrations of fluorene in the diet. One set of rats was exposed to several concentrations (number not specified) ranging from 0.062-1.0% fluorene in the diet for 104 days. These rats were maintained on diets with fluorene concentrations of 0.5 and 1.0%; they experienced significant decreases in their rate of growth, but in other aspects they appeared normal. The second set received either 0.125, 0.25 or 0.5% fluorene in the diet for 453 days.

One rat exposed to 0.125% fluorene in the diet developed a small benign kidney tubular adenoma. The total number of animals treated was not indicated, nor was a control group described.

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Fluorene produced no positive results in reverse mutation assays in five strains of *Salmonella typhimurium* (1000 ug/plate) or in forward mutation assays in *Salmonella* strain TM677 (50 ug/mL) (McCann et al., 1975; LaVoie et

al., 1979, 1981; Sakai et al., 1985; Bos et al., 1988; Kaden et al., 1979; Mamber et al., 1983). In a DNA damage assay using *S. typhimurium* TA1535, Nakamura et al. (1987) reported that fluorene at concentrations of up to 16.7 ug/mL was not positive. DNA damage assays with fluorene were not positive in *Escherichia coli* at concentrations of up to 2 mg/mL (Mamber et al., 1983, 1984) or in primary rat hepatocyte cultures at a maximum concentration of 3 mM (Sina et al., 1983). In a phage induction assay using *Escherichia coli* as a host, fluorene was not positive at concentrations of up to 1 mg/mL (Mamber et al., 1984).

In an unscheduled DNA synthesis assay the exposure of primary rat hepatocytes to 10 nmol and 100 nmol/mL fluorene did not yield positive results (Probst et al., 1981; Williams et al., 1989). Fluorene produced positive results in a DNA damage assay (strand-break assay) in L5178Y/mouse lymphoma cells at 0.15 uM in the presence of hepatic homogenates and at 0.5 uM in the absence of hepatic homogenates (Garberg et al., 1988). In forward mutation assays in L5178Y/mouse lymphoma cells, fluorene was not positive at concentrations of up to 30 and 60 ug/mL in the presence and absence of hepatic homogenates, respectively (Amacher et al., 1981; Oberly et al., 1984).

#### II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

None.

#### II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

None.

#### II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

##### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1990. Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons (PAHs). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. Final Draft. ECAO-CIN-D010.

##### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The 1990 Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons has received Agency and external review.

Agency Work Group Review: 02/07/90

Verification Date: 02/07/90

##### II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

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Option? TYPE 15/2

File 15; Entry 1; Accession No. 1457

(CAS) CAS Registry Number: 193-39-5

(MAT) Material Name: Indeno[1,2,3-cd]pyrene

(SYN) Synonyms:

Indeno(1,2,3-cd)pyrene;  
HSDB 5101;  
indeno(1,2,3-cd)pyrene;  
o-PHENYLENEPYRENE;  
RCRA WASTE NUMBER U137;  
1,10-(O-PHENYLENE)PYRENE;  
1,10-(1,2-Phenylene)pyrene;  
2,3-o-PHENYLENEPYRENE;  
2,3-PHENYLENEPYRENE

(UPD) Update Date: 12-01-90

(EFF) Effective Date: 07-01-91

(STAT) Status:

STATUS OF DATA FOR Indeno[1,2,3-cd]pyrene

File On-Line 12-01-90

Category (section)	Status	Last
Revised		
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Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	
12-01-90		
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	no data	
Supplementary Data (V.)	no data	

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(CAR) Carcinogenicity Assessment:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen

Basis -- Based on no human data and sufficient data from animal bioassays.

Indeno[1,2,3-cd]pyrene produced tumors in mice following lung implants,

subcutaneous injection and dermal exposure.

Indeno[1,2,3-cd]pyrene tested

positive in bacterial gene mutation assays.

II.A.2. HUMAN CARCINOGENICITY DATA

None. Although there are no human data that specifically link exposure to

indeno[1,2,3-cd]pyrene to human cancers, indeno[1,2,3-cd]pyrene is a component

of mixtures that have been associated with human cancer. These include coal

tar, soots, coke oven emissions and cigarette smoke (U.S. EPA, 1984, 1990;

IARC, 1984).

II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. In carcinogen bioassays indeno[1,2,3-cd]pyrene exposure

resulted in increased incidences of epidermoid carcinomas in a lung

implantation study (Deutsch-Wenzel et al., 1983), injection site sarcomas in a

subcutaneous injection assay (Lacassagne et al., 1963) and skin tumors in

dermal application studies (Hoffman and Wynder, 1966; Rice et al., 1985a,

1986).

In a lifetime implant study, 3-month-old female Osborne-Mendel rats

(35/group) received lung implants of indeno[1,2,3-cd]pyrene in 0.05 mL of a

1:1 (v:v) mixture of beeswax and trioctanoin (Deutsch-Wenzel et

al., 1983).

Rats received either 0.16 mg (0.65 mg/kg), 0.83 mg (3.4 mg/kg) or 4.15 mg (17 mg/kg) indeno[1,2,3-cd]pyrene. Controls consisted of an untreated group and a group receiving an implant of the vehicle. Median survival times in weeks were as follows: untreated controls, 118; vehicle controls, 104; low-dose, 116; mid-dose, 109; and high-dose, 92. Incidence of epidermoid carcinomas in the lung and thorax (combined) showed a statistically significant dose-related increase. The incidences were: untreated controls, 0/35; vehicle controls, 0/35; low-dose, 4/35 (11%); mid-dose, 8/35 (23%); and high-dose, 21/35 (60%).

Groups of male and female CD-1 mice (n=32) received intraperitoneal injections of indeno[1,2,3-cd]pyrene in dimethyl sulfoxide (DMSO) on days 1, 8 and 15 after birth (total dose = 580 ug/mouse) and were evaluated for tumors upon sacrifice at 52 weeks of age (LaVoie et al., 1987). One male mouse (1/11) developed a lung adenoma, no tumors occurred in female mice. Tumor incidence was not significantly different from vehicle controls. This test is considered to be a short-term lung tumor assay.

In mouse skin painting assays, indeno[1,2,3-cd]pyrene tested positive for cancer-initiating activity in several mouse strains (Hoffmann and Wynder, 1966; Rice et al., 1985a, 1986). In the Hoffmann and Wynder (1966) study female Swiss albino Ha/ICR/Mil mice (20/group) were given topical applications of indeno[1,2,3-cd]pyrene prepared as dioxane (at 0.05 and 0.1%) or in acetone solutions (at 0.01, 0.05 and 0.1%). Dioxane preparations did not induce skin tumors. By contrast, acetone solutions of indeno[1,2,3-cd]pyrene produced skin tumors in a dose-related fashion. No tumors were observed in animals painted with 0.01 or 0.05% indeno[1,2,3-cd]pyrene in acetone; 0.1% induced six papillomas and three carcinomas beginning at 9 months; and 0.5% resulted in seven papillomas and five carcinomas with the first tumor appearing at 3

months. The authors also reported that a total dose of 250 mg indeno[1,2,3-cd]pyrene delivered in 10 applications in 2 days was a sufficient initiating dose when followed by promotion with croton oil.

To examine the initiating capability of the compound's major metabolites in mouse skin, indeno[1,2,3-cd]pyrene was applied to the shaved backs of 20

Crl:CD-1(ICR)BR female mice (Rice et al., 1986). Acetone solutions were applied every other day for 10 days for a total initiating dose of 1 mg

indeno[1,2,3-cd]pyrene. This was followed 10 days later by applications of

the promotor tetradecanoylphorbol (TPA) (0.0025% in 100 mL acetone) 3

times/week for 20 weeks. Tumor incidence was essentially 100%. Indeno[1,2,3-

cd]pyrene-1,2-diol and -1,2-oxide treatment both resulted in 80% tumor

incidence in contrast to 8-hydroxy- and acetone-treated controls (approximately 25 and 5%, respectively).

An earlier initiation-promotion bioassay performed by Rice et al. (1985a)

showed a pronounced dose-response relationship for tumors. Following the same

protocol described above, an 80% tumor incidence was observed in mice

receiving a total initiating dose of 1 mg indeno[1,2,3-cd]pyrene with an

average of about four tumors/mouse after 22 weeks of promotion. However, when

the total initiating dose was decreased to 100 or 300 mg/mouse, the number of

tumor-bearing mice was not significantly increased.

Injection site sarcomas were reported in 10/14 male and 1/14 female

XVIIc/2 mice administered 3 injections at 1-month intervals of 0.6 mg

indeno[1,2,3-cd]pyrene. No concurrent controls appear to have been run in

this experiment; the authors report, however, that in this mouse strain no

spontaneous subcutaneous tumors have been reported (Lacassagne et al., 1963).

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Indeno[1,2,3-cd]pyrene produced positive results in reverse mutation assays in Salmonella typhimurium strains TA100 and TA98 (2-3 ug/plate) (LaVoie et al., 1979; Hermann et al., 1980; Rice et al., 1985b).

## II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1984. Carcinogen Assessment of Coke Oven Emissions. Office of Health and Environmental Assessment, Washington, DC. EPA 600/6-82-003F. NTIS PB 84-170181.

U.S. EPA. 1990. Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons (PAHs). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. Final Draft. ECAO-CIN-D010, September, 1990.

### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The 1990 Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons has received Agency and external review.

Agency Work Group Review: 02/07/90

Verification Date: 02/07/90

### II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

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Robert E. McGaughy / ORD -- (202)382-5889 / FTS 382-5889

Captured 6/11/92

1 - IRIS

IRSN - 271

DATE - 920604

UPDT - 06/04/92, 52 fields

STAT - Oral RfD Assessment (RDO) message 02/01/91

STAT - Inhalation RfC Assessment (RDI) no data

STAT - Carcinogenicity Assessment (CAR) on-line 05/01/91

STAT - Drinking Water Health Advisories (DWHA) no data

STAT - U.S. EPA Regulatory Actions (EXSR) on-line 06/01/92

IRH - 09/26/88 CAR Carcinogen summary on-line

IRH - 02/01/89 MCLG Effect level corrected in discussion

IRH - 06/01/89 CARDR Primary contact changed

IRH - 06/01/89 CAA Reference corrected - changed number for part in CFR

IRH - 12/01/89 CAREV Last paragraph - Correct Van Esch 1969 citation

IRH - 12/01/89 REFS Bibliography on-line

IRH - 07/01/90 RDO Changed contact J. Cohen's office and telephone number

IRH - 07/01/90 RCRA EPA contact changed

IRH - 02/01/91 RDO Message revised to include new EPA document

IRH - 02/01/91 RDO EPA contacts changed

IRH - 05/01/91 CAREV Text edited

IRH - 01/01/92 EXSR Regulatory actions updated

IRH - 06/01/92 MCL MCL monitoring reqs. and BAT corrected

RLEN - 18586

NAME - Lead and compounds (inorganic)

RN - 7439-92-1

SY - Lead

SY - Lead and compounds

SY - plumbum

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RDO -

o ORAL RfD SUMMARY :

A great deal of information on the health effects of lead has been obtained through decades of medical observation and scientific research. This information has been assessed in the development of air and water quality criteria by the Agency's Office of Health and Environmental Assessment (OHEA) in support of regulatory decision-making by the Office of Air Quality Planning and Standards (OAQPS) and by the Office of Drinking Water (ODW). By comparison to most other environmental toxicants, the degree of uncertainty about the health effects of lead is quite low. It appears that some of these effects, particularly changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development, may occur at blood lead levels so low as to be essentially without a threshold. The Agency's RfD Work Group discussed inorganic lead (and lead compounds) at two meetings (07/08/85 and 07/22/85) and considered it inappropriate to develop an RfD for inorganic lead. For additional information, interested parties are referred to the 1986 Air Quality Criteria for Lead (EPA-600/8-83/028a-dF) and its 1990 Supplement (EPA/600/8-89/049F) or the following Agency scientists:

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J. Michael Davis / ORD - (919)541-4162 / FTS 629-4162

Jeff Cohen / ODW -- (202)260-5456 / FTS 260-5456

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**CAREV-**

- o **CLASSIFICATION** : B2; probable human carcinogen
- o **BASIS FOR CLASSIFICATION** : Sufficient animal evidence. Ten rat bioassays and one mouse assay have shown statistically significant increases in renal tumors with dietary and subcutaneous exposure to several soluble lead salts. Animal assays provide reproducible results in several laboratories, in multiple rat strains with some evidence of multiple tumor sites. Short term studies show that lead affects gene expression. Human evidence is inadequate.
- o **HUMAN CARCINOGENICITY DATA :**

Inadequate. There are four epidemiologic studies of occupational cohorts exposed to lead and lead compounds. Two studies (Dingwall-Fordyce and Lane, 1963; Nelson et al., 1982) did not find any association between exposure and cancer mortality. Selevan et al. (1985), in their retrospective cohort mortality study of primary lead smelter workers, found a slight decrease in the total cancer mortality (SMR=95). Apparent excesses were observed for respiratory cancer (SMR=111, obs=41,  $p>0.05$ ) and kidney cancer (SMR=204, obs=6,  $p>0.05$ ). Cooper and Gaffey (1975) and Cooper (1985 update) performed a cohort mortality study of battery plant workers and lead smelter workers. They found statistically significant excesses for total cancer mortality (SMR=113, obs=344), stomach cancer (SMR=168, obs=34), and lung cancer (SMR=124, obs=109) in the battery plant workers. Although similar excesses were observed in the smelter workers, they were not statistically significant. Cooper and Gaffey (1975) felt it was possible that individual subjects were monitored primarily on the basis of obvious signs of lead exposure, while others who showed no symptoms of lead poisoning were not monitored.

All of the available studies lacked quantitative exposure information, as well as information on the possible contribution from smoking. All studies also included exposures to other metals such as arsenic, cadmium, and zinc for which no adjustment was done. The cancer excesses observed in the lung and stomach were relatively small ( $<200$ ). There was no consistency of site among the various studies, and no study showed any dose-response relationship. Thus, the available human evidence is considered to be inadequate to refute or demonstrate any potential carcinogenicity for humans from lead exposure.

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o **ANIMAL CARCINOGENICITY DATA :**

Sufficient. The carcinogenic potential of lead salts (primarily phosphates and acetates) administered via the oral route or by injection has been demonstrated in rats and mice by more than 10 investigators. The most characteristic cancer response is bilateral renal carcinoma. Rats given lead

acetate or subacetate orally have developed gliomas, and lead subacetate also produced lung adenomas in mice after i.p. administration. Most of these investigations found a carcinogenic response only at the highest dose. The lead compounds tested in animals are almost all soluble salts. Metallic lead, lead oxide and lead tetraalkyls have not been tested adequately. Studies of inhalation exposure have not been located in the literature.

Azar et al. (1973) administered 10, 50, 100, and 500 ppm lead as lead acetate in dietary concentrations to 50 rats/sex/group for 2 years. Control rats (100/sex) received the basal laboratory diet. In a second 2-year feeding study, 20 rats/group were given diets containing 0, 1000, and 2000 ppm lead as lead acetate. No renal tumors were reported in the control groups or in treated animals of either sex receiving 10 to 100 ppm. Male rats fed 500, 1000, and 2000 ppm lead acetate had an increased renal tumor incidence of 5/50, 10/20, and 16/20, while 7/20 females in the 2000-ppm group developed renal tumors.

The Azar et al. (1973) study is limited by the lack of experimental detail. The possibility of environmental contamination from lead in the air or drinking water was not mentioned. The strains of rats used were not specified in the study, but the Health Effects Assessment for Lead (U.S. EPA, 1984) indicates the rats were Wistar strain. The weight gain at 1000 and 2000 ppm was reported to be depressed, but details were not given.

Kasprzak et al. (1985), in investigating the interaction of dietary calcium on lead carcinogenicity, fed 1% lead subacetate (8500 ppm Pb) to male Sprague-Dawley rats in the diet for 79 weeks. Of the rats surviving (29/30) in this treatment group beyond 58 weeks, 44.8% had renal tumors. Four rats had adenocarcinomas; the remaining nine had adenomas. Bilateral tumors were noted. No renal tumors were noted among the controls.

As part of a study to determine interactions between sodium nitrite, ethyl urea and lead, male Sprague-Dawley rats were given lead acetate in their drinking water for 76 weeks (Koller et al., 1986). The concentration of lead was 2600 ppm. No kidney tumors were detected among the 10 control rats. Thirteen of 16 (81%) lead-treated rats had renal tubular carcinoma; three tumors were detected at 72 weeks and the remainder detected at the termination of the study.

Van Esch and Kroes (1969) fed basic lead acetate at 0, 0.1%, and 1.0% in the diet to 25 Swiss mice/sex/group for 2 years. No renal tumors developed in the control group, but 6/25 male mice of 0.1% basic lead acetate group had renal tumors (adenomas and carcinomas combined). In the 1.0% group, one female had a renal tumor. The authors thought that the low incidence in the 1.0% group was due to early mortality.

Hamsters given lead subacetate at 0.5% and 1% in the diet had no significant renal tumor response (Van Esch and Kroes, 1969).

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#### o SUPPORTING DATA :

Lead acetate induces cell transformation in Syrian hamster embryo cells (DiPaolo et al., 1978) and also enhances the incidence of simian adenovirus induction. Lead oxide showed similar enhanced adenovirus induction (Casto et

al., 1979).

Under certain conditions lead compounds are capable of inducing chromosomal aberrations in vivo and in tissue cultures. Grandjean et al. (1983) showed a relationship between SCE and lead exposure in exposed workers. Lead has been shown, in a number of DNA structure and function assays, to affect the molecular processes associated with the regulation of gene expression (U.S. EPA, 1986).

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**CARDR-**  
**o CARCINOGENICITY SOURCE :**

U.S. EPA. 1984. Health Effects Assessment for Lead. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Emergency and Remedial Response, Washington, DC. EPA/540/1-86/055. NTIS PB85-163996/AS.

U.S. EPA. 1986. Air Quality Criteria Document for Lead. Volumes III, IV. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC, for the Office of Air Quality Planning and Standards. EPA-600/8-83/028dF.

U.S. EPA. 1987. Preliminary review of the carcinogenic potential of lead associated with oral exposure. Prepared by the Office of Health and Environmental Assessment, Carcinogenic Assessment Group, Washington DC, for the Office of Drinking Water, Office of Solid Waste and the Office of Emergency and Remedial Response (Superfund). OHEA-C-267. Internal Review Draft.

The review of the carcinogenic potential of lead associated with oral exposure has received Agency review.

The 1986 Air Quality Criteria Document for Lead has received Agency and External Review.

**DOCUMENT**

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o REVIEW DATES : 05/04/88  
o VERIFICATION DATE : 05/04/88  
o EPA CONTACTS :

William Pepelko / ORD -- (202)260-5898 / FTS 260-5898

James Cogliano / ORD -- (202)260-9243 / FTS 260-9243

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CAA -

Considers technological or economic feasibility? -- No



Discussion -- Under Section 109 of the CAA, EPA has set a primary (health-based) NAAQS for lead of 1.5 ug/cu.m, calendar quarter average not to be exceeded (43 FR 41258, 10/05/78). The secondary (welfare-based) NAAQS is identical to the primary standard. EPA is currently reviewing these standards to determine if changes are warranted.

Reference -- 40 CFR 50.12

U.S. EPA Contact -- Air Quality Management Division / OAQPS /  
(919)541-5656 / FTS 629-5656

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WQCHU-

Water and Fish Consumption --  $5.0E+1$  ug/L

Fish Consumption Only -- None

Considers technological or economic feasibility? -- NO

Discussion -- The criterion was set at the existing drinking water standard in 1980.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS  
(202)260-1315 / FTS 260-1315

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WQCAQ-

Freshwater:

Acute --  $8.2E+1$  ug/L (1-hour average)

Chronic --  $3.2E+0$  ug/L (4-day average)

Marine:

Acute --  $1.40E+2$  ug/L (1-hour average)

Chronic --  $5.6E+0$  ug/L (4-day average)

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute and chronic tests on a variety of species. The toxicity of this compound in freshwater is hardness dependent. The values given are for a hardness of 100 mg/L CaCO<sub>3</sub>. For a more complete discussion, see the referenced notice.

Reference -- 50 FR 30784 (07/29/85)

EPA Contact -- Criteria and Standards Division / OWRS  
(202)260-1315 / FTS 260-1315

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**MCLG -**

Value (status) -- 0 mg/L (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion -- The MCLG for lead is zero based on (1) occurrence of low level effects and difficulties in identifying clear threshold levels, (2) the overall Agency goal of reducing total lead exposures, and (3) the classification of lead as a group B2 carcinogen.

Reference -- 56 FR 26460 (06/07/91); 56 FR 32112 (07/15/91)

EPA Contact -- Health and Ecological Criteria Division / OST /  
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

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**MCL -**

Value -- None (Final, 1991)

Considers technological or economic feasibility? -- YES

Discussion -- EPA concluded that setting an MCL for lead is not feasible and believes that the treatment approach contained in the final rule (corrosion control, source water reduction, public education and lead service line problems associated with establishing MCL's.

Monitoring requirements -- Tap water monitoring for lead and copper to determine whether a system is subject to the treatment technique requirements. Water quality parameter sampling to determine the effectiveness of optional corrosion control treatment. Source water monitoring for lead and copper to determine source water's contribution to total tap water lead and copper levels, and the need for treatment. Monitoring schedules vary by system size and type of monitoring.

Analytical methodology -- Atomic absorption/furnace technique (EPA 239.2; ASTM D-3559-85D; SM 3113); inductively-coupled plasma/mass spectrometry (EPA 200.8); atomic absorption/platform furnace technique (EPA 200.9).

Best available technology --

Optimal corrosion control treatment: pH/alkalinity adjustment, calcium adjustment; addition of corrosion inhibitor.

Source water treatment: Coagulation/filtration; ion exchange; lime softening; reverse osmosis.

Public education.

Lead service line replacement.

Reference -- 45 FR 57332 (08/27/80); 53 FR 31517 (08/18/88); 56 FR 26460 (06/07/91); 56 FR 32112 (07/15/91).

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

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CERC -

Value (status) -- 1 pound (Statutory, 1987)

Considers technological or economic feasibility? -- NO

Discussion -- The statutory 1-pound RQ for lead is retained pending assessment of its potential carcinogenicity and may be adjusted in a future notice of proposed rulemaking when the evaluation of available data is completed. Lead was evaluated for chronic toxicity, but was not ranked for toxicity because of insufficient data.

Reference -- 52 FR 8140 (03/16/87); 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)260-3000 / FTS 260-3000  
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RCRA -

Status -- Listed (total lead)

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)260-3000 / FTS 260-3000

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TSCA -

No data available

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OREF - None

IREF - None

CREF - Anderson, E.L., and CAG (Carcinogenic Assessment Group). 1983. Quantitative approaches in use to assess cancer risk. *Risk Analysis*. 3: 277-295.

CREF - Azar, A., H.J. Trochimowicz and M.E. Maxfield. 1973. Review of lead studies in animals carried out at Haskell Laboratory - Two year feeding study and response to hemorrhage study. In: Barth D., A. Berlin, R. Engel, P. Recht and J. Smeets, Ed. *Environmental health aspects of lead: Proceedings International Symposium; October 1972; Amsterdam, The Netherlands. Commission of the European Communities, Luxembourg.* p. 199-208.

CREF - Casto, B.C., J. Meyers and J.A. DiPaolo. 1979. Enhancement of viral transformation for evaluation of the carcinogenic or mutagenic potential of inorganic metal salts. *Cancer Res.* 39: 193-198.

CREF - Cooper, W.C. 1985. Mortality among employees of lead battery plants and lead producing plants, 1947-1980. *Scand. J. Work Environ. Health*. 11: 331-345.

CREF - Cooper, W.C. and W.R. Gaffey. 1975. Mortality of lead workers. In: *Proceedings of the 1974 Conference on Standards of Occupational Lead Exposure*, J.F. Cole, Ed., February, 1974. Washington, DC. *J. Occup. Med.* 17: 100-107.

CREF - Dingwall-Fordyce, I. and R.E. Lane. 1963. A follow-up study of lead workers. *Br. J. Ind. Med.* 20: 313-315.

CREF - DiPaolo, J.A., R.L. Nelson and B.C. Casto. 1978. In vitro neoplastic transformation of Syrian hamster cells by lead acetate and its relevance to environmental carcinogenesis. *Br. J. Cancer*. 38: 452-455.

CREF - Grandjean, P., H.C. Wulf and E. Niebuhr. 1983. Sister chromatid exchange in response to variations in occupational lead exposure. *Environ. Res.* 32: 199-204.

CREF - Kasprzak, K.S., K.L. Hoover and L.A. Poirier. 1985. Effects of dietary calcium acetate on lead subacetate carcinogenicity in kidneys of male Sprague-Dawley rats. *Carcinogenesis*. 6(2): 279-282.

CREF - Koller, L.D., N.I. Kerkvliet and J.H. Exon. 1986. Neoplasia induced in male rats fed lead acetate, ethyl urea and sodium nitrate. *Toxicol. Pathol.* 13: 50-57.

CREF - Nelson, D.J., L. Kiremidjian-Schumacher and G. Stotzky. 1982. Effects of cadmium, lead, and zinc on macrophage-mediated cytotoxicity toward tumor cells. *Environ. Res.* 28: 154-163.

CREF - Selevan, S.G., P.J. Landrigan, F.B. Stern and J.H. Jones. 1985. Mortality of lead smelter workers. *Am. J. Epidemiol.* 122: 673-683.

CREF - Van Esch, G.J. and R. Kroes. 1969. The induction of renal tumors by feeding of basic lead acetate to mice and hamsters. *Br. J. Cancer*. 23: 265-271.

**CREF - U.S. EPA. 1984. Health Effects Assessment for Lead. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Emergency and Remedial Response, Washington, DC. EPA/540/1-86/055. NTIS PB85-163996/AS.**

**CREF - U.S. EPA. 1986. Air Quality Criteria Document for Lead. Volumes III, IV. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC, for the Office of Air Quality Planning and Standards. EPA-600/8-83/028dF.**

**CREF - U.S. EPA. 1987. Preliminary review of the carcinogenic potential of lead associated with oral exposure. Prepared by the Office of Health and Environmental Assessment, Carcinogenic Assessment Group, Washington DC, for the Office of Drinking Water, Office of Solid Waste and the Office of Emergency and Remedial Response (Superfund). OHEA-C-267. Internal Review Draft.**

**HAREF- None**

TYPE 11/2

File 11; Entry 1; Accession No. 1373

(CAS) CAS Registry Number: 7439-96-5

(MAT) Material Name: Manganese

(SYN) Synonyms:  
COLLOIDAL MANGANESE;  
MAGNACAT;  
MANGAN;  
Manganese;  
MANGAN NITRIDOVANY;  
TRONAMANG

(UPD) Update Date: 12-06-90

(EFF) Effective Date: 07-01-91

(STAT) Status:

STATUS OF DATA FOR Manganese

File On-Line 09-26-88

Category (section) -----	Status -----	Last Revised -----
Oral RfD Assessment (I.A.)	on-line	08-01-90
Inhalation RfC Assessment (I.B.)	on-line	12-06-90
Carcinogenicity Assessment (II.)	on-line	08-01-90
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	no data	
Supplementary Data (V.)	no data	

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(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS  
I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

### I.A.1. ORAL RFD SUMMARY

Critical Effect -----	Experimental Doses* -----	UF -----	MF ---	RFD -----
CNS effects	NOAEL: 0.14 mg/kg/day	1	1	1E-1 mg/kg/day

Human Chronic  
Ingestion Data

LOAEL: None

WHO, 1973;  
Schroeder et al., 1966;  
NRC, 1989

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\*Conversion Factors: The NOAEL of 10 mg/day (0.14 mg/kg/day for 70 kg adult) for chronic human consumption of manganese is based on a composite of data from the above three references. WHO (1973) reported no adverse effects in humans consuming supplements of 8-9 mg Mn/day (0.11-0.13 mg/kg/day). Schroeder et al. (1966) reported a chronic human NOAEL OF 11.5 mg Mn/day (0.16 mg/kg/day). The NRC (1989) determined "safe and adequate" levels to be 2-5 mg Mn/day for adults (0.03-0.07 mg/kg/day). It is important to recognize that manganese is an essential element in human nutrition. It is also important to recognize that this oral RFD is based on a total dietary intake, and this amount of manganese is not necessarily acceptable if the intake were from drinking water alone. This difference is due to the fact that manganese in drinking water is more bioavailable than manganese in food.

### I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RFD)

WHO (World Health Organization). 1973. Trace elements in human nutrition: Manganese. Report of a WHO Expert Committee. Technical Report Service, 532, WHO, Geneva, Switzerland. p. 34-36.

Schroeder, H.A., D.D. Balassa and I.H. Tiptr. 1966. Essential trace metals in man: Manganese, a study in homeostasis. J. Chron. Dis. 19: 545-571.

NRC (National Research Council). 1989. Recommended Dietary Allowances, 10th ed. Food and Nutrition Board, National Research Council, National Academy

Press, Washington, DC. p. 230-235.

The World Health Organization (WHO, 1973) reviewed several investigations of adult diets and reported the average daily consumption of manganese to range from 2.0 to 8.8 mg Mn/day. Higher manganese intakes are associated with diets high in whole cereals, nuts, green leafy vegetables, and tea. From manganese balance studies, the WHO concluded that 2 to 3 mg/day is adequate for adults and 8 to 9 mg/day is "perfectly safe."

Evaluations of standard diets from the United States, England, and Holland reveal average daily intakes of 2.3 to 8.8 mg Mn/day (Schroeder et al., 1966). However, depending on individual diets, a normal intake may be even higher, especially from a vegetarian diet. These levels are considered to be safe for chronic human ingestion.

No signs of toxicity were reported in patients (number not specified) given 30 mg manganese citrate (9 mg Mn/day) for many months. Assuming the patients also consumed 2.5 mg Mn/day in food, the total manganese intake would be approximately 11.5 mg Mn/day.

The Food and Nutrition Board of the National Research Council (NRC, 1989) determined an "adequate and safe" intake of manganese to be 2 to 5 mg/day for adults. This level was chosen because it includes an "extra margin of safety" from the level of 10 mg/day, which can be considered to be safe.

#### I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF - 1. The information used to determine the oral RfD for manganese was taken from many large populations. Humans exert an efficient homeostatic control over manganese such that body burdens are kept constant with variations in diet. There are no subpopulations which are believed to be more sensitive to manganese at this level. The use of an uncertainty factor of 1 is supported by the fact that manganese is an essential element, being required for normal human growth and maintenance of health. It has also been suggested that children are less susceptible to manganese intoxication and may require slightly higher levels of manganese than do adults.

MF - 1.

#### I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

A small-scale epidemiologic study of manganese in drinking water was performed by Kondakis et al. (1989). Three areas in northwest Greece were chosen for this study, with manganese concentrations of 3.6 to 14.6 ug/L in



area A, 81.6 to 252.6 ug/L in area B, and 1600 to 2300 ug/L in area C. The study included only individuals over the age of 50 drawn from a random sample of 10% of all households (n=62, 49 and 77 for areas A, B, and C, respectively). The authors reported that "all areas were similar with respect to social and dietary characteristics," but few details were reported. The amount of manganese in the diet was not reported. The individuals chosen were submitted to a neurological examination, the score of which represents a composite of the presence and severity of 33 symptoms (e.g., weakness/fatigue, gait disturbances, tremors, dystonia). Whole blood and hair manganese concentrations were also determined. The mean concentration of manganese in hair was 3.51, 4.49, and 10.99 ug/g dry weight for areas A, B, and C, respectively ( $p < 0.0001$  for area C vs. A). However, the concentration of manganese in whole blood did not differ between the three areas. The mean ( $\bar{x}$ ) and range ( $r$ ) of neurologic scores were as follows: Area A (males:  $\bar{x}=2.4$ ,  $r=0-21$ ; females:  $\bar{x}=3.0$ ,  $r=0-18$ ; both:  $\bar{x}=2.7$ ,  $r=0-21$ ); Area B (males:  $\bar{x}=1.6$ ,  $r=0-6$ ; females:  $\bar{x}=5.7$ ,  $r=0-43$ ; both:  $\bar{x}=3.9$ ,  $r=0-43$ ); Area C (males:  $\bar{x}=4.9$ ,  $r=0-29$ ; females:  $\bar{x}=5.5$ ,  $r=0-21$ ; both:  $\bar{x}=5.2$ ,  $r=0-29$ ). While there appears to be an increasing trend in the neurological scores, this data should be interpreted with caution. The authors did not provide any individual data, and the large range for females in area B indicates that a single outlier may have been responsible for the increased mean. The mean score for men in area B was actually lower than that in area A. The authors indicate that the difference in mean scores for area C vs. A was significantly increased (Mann-Whitney  $z=3.16$ ,  $p=0.002$  for both sexes combined). While this finding should be acknowledged, its significance, particularly with regard to the concentration of manganese in drinking water, is questionable. This study has several flaws, most notably: 1) the small number of individuals tested; 2) the lack of scatter data; 3) the lack of information provided on social and other dietary and drinking water factors; 4) this study may not have been truly unbiased because the examining neurologists were listed as authors of the paper. In summary, this study raises some questions about acceptable levels of manganese in drinking water, but is inadequate to serve as the basis for a separate water RfD. It may, however, serve to caution risk assessors against using a total oral RfD (based principally on dietary intake) to establish an

acceptable drinking water concentration, without taking into consideration issues such as differential absorption.

A report by Kawamura et al. (1941) described toxicologic responses in humans consuming large amounts of manganese dissolved in drinking water. The source of the manganese came from about 400 dry-cell batteries which were buried near a drinking water well. Sixteen cases of manganese poisoning were reported, with symptoms including lethargy, increased muscle tonus, tremor, and mental disturbances. The most severe symptoms were seen in elderly people, with children being affected to a lesser degree. Three individuals died, one from suicide. The cause of death for the other two was not reported, but the autopsy of one individual revealed manganese concentration in the liver to be 2 to 3 times higher than controls. Zinc levels were also increased in the liver. The well water was not analyzed until 1 month after the outbreak, at which time it contained approximately 14 mg Mn/L. However, when re-analyzed 1 month later, the levels were decreased by about half. Therefore, by retrospective extrapolation, the concentration of manganese at the time of exposure was probably at least 28 mg Mn/L. Assuming an adult body weight of 70 kg and a water consumption of 2 L/day, this would be equivalent to an intake of 0.8 mg Mn/kg bw/day from drinking water alone.

While there is little information concerning manganese poisoning in humans by the oral route, there is a well-documented association of prolonged inhalation of manganese dusts with psychological and neurological disorders.

Several toxicity studies on manganese have been performed in laboratory animals. Most of these have been inhalation studies, demonstrating an effect on both the brain and lungs. Several oral studies have been performed in rodents that demonstrated biochemical changes in the brain following administration of 1 mg  $\text{MnCl}_2 \cdot 4\text{H}_2\text{O}$ /mL in drinking water (approximately 38.9 mg Mn/kg bw/day) (Lai et al., 1981, 1982; Laung et al., 1981). However, rodents do not exhibit the same neurological deficits that humans do following exposure to manganese, so the relevance of these biochemical changes has been challenged. While primates are considered to be the species of choice for modeling the human response to manganese poisoning, only one limited oral study

has been performed in a group of four rhesus monkeys (Gupta et al., 1980).

Muscular weakness and rigidity of the lower limbs developed after 18 months of exposure to 6.9 mg Mn/kg bw/day (as MnCl<sub>2</sub>·4H<sub>2</sub>O). Histological analysis showed degenerated neurons in the substantia nigra and scanty neuromelanin granules

in some of the pigmented cells.

#### I.A.5. CONFIDENCE IN THE ORAL RfD

Study: High

Data Base: Medium

RfD: Medium

Many studies have reported similar findings with regard to the normal intake of manganese by humans. These data are considered to be superior to any data obtained from animal toxicity studies, especially as the physiologic requirements for manganese vary quite a bit among different species, with man requiring less than rodents (Schroeder et al., 1966).

It is again emphasized that this oral RfD is based on a total dietary intake of manganese and is not intended to be applied directly to drinking water.

#### I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- This assessment is not presented in any existing U.S. EPA document.

Agency RfD Work Group Review: 05/17/90; 06/21/90

Verification Date: 06/21/90

#### I.A.7. EPA CONTACTS (ORAL RfD)

Sue Velazquez / ORD -- (513)569-7571 / FTS 684-7571

Julie Du / ODW -- (202)382-7583 / FTS 382-7588

### I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

#### I.B.1. INHALATION RfC SUMMARY

Critical Effect	Exposures*	UF	MF	RfC
-----	-----	-----	---	-----
Increased prevalence	NOEL: None	300	3	4E-4

of respiratory

mg/cu.m

symptoms and psycho-  
motor disturbances

LOAEL: 0.97 mg/cu.m  
LOAEL(ADJ): 0.34 mg/cu.m  
LOAEL(HEC): 0.34 mg/cu.m

Occupational exposure  
to inorganic manganese

Roels et al., 1987

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\*Conversion Factors: The LOAEL is based on an 8-hour TWA occupational exposure. The TWA of total airborne manganese dust ranged from 0.07-8.61

mg/cu.m, and the median was 0.97 mg/cu.m. This is a respiratory and extrarrespiratory effect of a particle exposure. MVho = 10 cu.m./day, MVh =

20 cu.m/day. LOAEL(HEC) = 0.97 mg/cu.m x (MVho/MVh) x 5 days/7days = 0.34 mg/cu.m.

#### I.B.2. PRINCIPAL AND SUPPORTING STUDIES (INHALATION RFC)

Roels, H., R. Lauwerys, J-P. Buchet et al. 1987. Epidemiological survey among workers exposed to manganese: Effects on lung, central nervous system, and some biological indices. Am. J. Ind. Med. 11: 307-327.

Roels et al. (1987) conducted a cross-sectional study in 141 male workers exposed to manganese dioxide, tetroxide and various salts (sulfate, carbonate and nitrate). A matched group of 104 male workers was selected as a control group. The two groups were matched for socioeconomic status and background environmental factors; in addition, both groups had comparable workload and workshift characteristics. The TWA of total airborne manganese dust ranged from 0.07-8.61 mg/cu.m, respectively, with an overall mean and median of 1.33 and 0.97 mg/cu.m. The authors noted that there was an increase in production between 1965 (440 metric tons) and 1981 (22,000 metric tons) and presumably exposure with time. Thus exposure, particularly for individuals with long employment durations, may have been lower. The duration of employment ranged from 1-19 years with a mean of 7.1 years. The particle size and purity of the dust were not reported. Neurological examination, psychomotor tests (simple reaction time, short-term memory and hand tremor), lung function test (forced

vital capacity, forced expiratory volume, peak expiratory flow rate and maximal expiratory flow rate at 50 and 75% of the FVC), blood and urine tests and a questionnaire were used to assess possible toxic effects of manganese exposure. The questionnaire was designed to detect CNS and respiratory symptoms.

Concentration-response relationships between length of exposure or urinary manganese levels and the prevalence of abnormal CNS findings were not observed. A significantly higher prevalence of coughs during the cold season, dyspnea during exercise and recent episodes of acute bronchitis were found in the exposed group. Lung ventilatory parameters were mildly altered in the exposed smokers. Significant alterations were found in simple reaction time (visual), audioverbal short-term memory test, eye-hand coordination, and hand steadiness test in the workers exposed to manganese. In general, this study is adequate to derive a risk assessment, however, certain limitations should be noted. One shortcoming is the lack of adjustment for age in the psychomotor measures. Age-standardization was used in the short-term memory task, but not in the measures of reaction time and tremor (hand steadiness and eye-hand coordination). However, since the mean age of the control group was higher than that of the exposed group, the likely effect of a lack of age adjustment is to underestimate the effect of manganese. Another limitation of the Roels et al. study was the lack of correction for multiple tests. Differences between control and exposed groups on several neurobehavioral measures were assessed with simple t tests or chi-square tests. With alpha = 0.05, one in twenty such tests could be found statistically significant by chance alone. However, it appears that this percentage was well exceeded, e.g., 5 or 8 reaction time measures were significant and 7 of 11 short-term memory measures were significant. Thus, these flaws in the Roels et al., study do not appear to compromise its utility for risk assessment purposes. Based upon the increased psychomotor disturbances, a LOAEL of 0.97 mg/cu.m was identified [where the LOAEL(HEC) = 0.34 mg/cu.m].

Chandra et al. (1981) examined 60 welders from three separate plants (20/plant) exposed to manganese fumes. A matched control group of 20 workers was also examined. The average length of employment in plant 1 was >10 years.

with a manganese level of 0.24-0.99 mg/cu.m (mean = 0.31 cu.m) in the breathing zone. In plant 2, the air concentration was 0.50-0.80 mg/cu.m (mean = 0.57 cu.m), and the length of exposure ranged from 2 years to more than 20 years. In plant 3, half of the workers were employed for less than 10 years and the other half for more than 15 years. The air concentration of manganese was 0.88-2.6 mg/cu.m (mean = 1.74 cu.m). The manganese compounds and the presence of other compounds in the fumes were not reported. In plant 1, the workers complained of frequent occurrence of colds, cough and short hyperpyrexia. The workers of all three plants often reported insomnia. No other subjective effects were reported by the workers in plants 2 and 3. No hematological alterations were observed in hemoglobin, RBC and WBC counts. Positive neurological signs (brisk, deep reflexes in the legs and/or arms) were observed in 25, 50 and 45% of workers in plants 1, 2 and 3, respectively. Tremors were also observed in one and four workers in plants 1 and 2, respectively. No positive neurological signs were observed in the control workers. Although significant effects are reported for "deep reflexes" and "tremors," it appears that these endpoints were assessed through a non-blind neurological examination. If in fact the examiner was aware of a subject's exposure condition, then the results are questionable. One may also question the sensitivity of a clinical neurological examination for detecting what could be quite subtle neurotoxic examination for detecting what could be quite subtle neurotoxic effects. The findings of Chandra et al., may be viewed as supportive. Increased serum calcium levels and urinary manganese levels were also observed in the welders. The calculated LOAEL(HEC) from the mean exposure of plant 1 is 0.11 mg/cu.m.

Personnel that were most exposed were selected from two Swedish foundries (15 from each plant) for inclusion in a study reported by Iregren (1990). The exposure to manganese varied from 0.02-1.4 mg/cu.m (mean=0.25 mg/cu.m) for 1-35 years (mean=9.9 years). Earlier exposure measurements made in both factories indicated that there were essentially no changes in either factory for the past 18 years. Exposed workers were matched to two workers not exposed to manganese from other industries for age, geographical area and type of work. Evaluation for neurobehavioral function was assessed by 8 computerized tests from the Swedish Performance Evaluation System and 2 manual

dexterity tests. After further adjustment for general cognitive abilities, there was significant difference between exposed and control groups for simple reaction time, the standard deviation of reaction time, and finger tapping speed of the dominant hand. The neurobehavioral differences between the two groups remained statistically significant even when verbal test scores were used as a covariate. The size of the reference group was reduced to 30 workers when controlled for apparent differences in cognitive abilities (the number of exposed workers did not change). No significant correlation was found within the exposed group to establish a concentration-response relationship. The LOAEL(HEC) is therefore calculated to be 0.09 mg/cu.m. This study, along with Chandra et al. (1981), provide a pattern of neurobehavioral effects of low-level occupational manganese exposure consistent with that reported by Roels et al., 1987.

#### I.B.3. UNCERTAINTY AND MODIFYING FACTORS (INHALATION RfC)

UF = 300. An uncertainty factor of 100 reflects 10 to protect sensitive individuals and 10 for use of a LOAEL. An additional factor of 3 was used to account for the less than chronic period of exposure.

MF = 3. A modifying factor of 3 is used to account for the uncertainty of exposure to manganese in the principal study. During the exposure period there was an exponential increase in manganese usage and production without accompanying changes in the plant processing area. It is therefore assumed that ambient manganese concentration during the exposure period was lower than that measured at the time of evaluation of neurological symptoms. Since the exposure cannot be quantitatively evaluated, a modifying factor is used.

#### I.B.4. ADDITIONAL STUDIES / COMMENTS (INHALATION RfC)

Manganese toxicity can vary depending upon the route of exposure. When ingested, manganese is considered to be among the least toxic of the trace elements. In the normal adult, between 3 and 10% of dietary manganese is absorbed. Total body stores are then regulated by a complex homeostatic mechanism involving absorption and excretion. Certain sub-populations such as the elderly, children, pregnant women and iron-deficient individuals may have increased absorption or altered clearance mechanisms resulting in an increased potential for excess total body manganese. As detailed in I.A.3. and I.A.4., toxicity from ingested manganese is rarely observed.

Understanding the inhalation toxicity of manganese requires consideration of particle dosimetry and subsequent pharmacokinetic events. Particle size will determine the site of deposition in the respiratory tract. Generally, in humans, fine mode particles (<2.5 microns) preferentially deposit in the pulmonary region and coarse mode particles (>2.5 microns) deposit in the tracheobronchial and extrathoracic regions. Those particles depositing in the extrathoracic and tracheobronchial regions are predominantly cleared by the mucociliary escalator into the gastrointestinal tract where absorption will be quite low (about 3%). For manganese, another possibility exists. A brief report (Perl and Good, 1987) suggested that another heavy metal, aluminum, was directly transported to the brain via nasal olfactory pathways (i.e., from extrathoracic deposition). One could speculate that this pathway may operate for manganese, raising additional difficulty in understanding target site dosage. Particles deposited in the pulmonary region will be cleared predominantly to the systemic compartment by absorption into the blood and lymph circulation. From all these factors, we assume 100% absorption of particles deposited in the pulmonary region, recognizing that this ignores other mechanisms that are likely to occur to some unquantified degree.

Unfortunately, the health effects data base on manganese does not include inhalation pharmacokinetics on the major oxides of manganese and the critical occupational studies did not measure particle size or speciate the manganese oxides involved. This prevents quantitative determinations of the dose delivered to the respiratory tract and estimates of target site doses. There are no quantitative data on the inhalation absorption rates of the different manganese compounds (U.S. EPA, 1984). Mena et al. (1969) observed no difference between the absorption of 1 micron particles of  $MnCl_2$  and  $Mn_2O_3$ . However, following intratracheal instillation of  $MnCl_2$  and  $Mn_3O_4$ , the chloride cleared 4 times faster than the oxide from the lung. After 2 weeks, similar levels of manganese remained in the lung (Drown et al., 1986). In general, respiratory clearance is faster for chemicals with greater water solubility. The potential for respiratory system toxicity typically is less for chemicals with faster clearance. However, water solubility cannot always predict respiratory tract clearance since cellular mechanisms can remove relatively



insoluble particles from the respiratory tract as rapidly as chemical dissolution and absorption. Faster brain manganese elimination half-times

have been reported in monkeys exposed to manganese via intravenous or subcutaneous injection (Newland et al., 1987). It is suggested that the slower rates of decline in brain manganese following inhalation probably reflects replenishment from manganese deposited in other organs, particularly the respiratory system. The issue is further confounded since an RfC could only be developed for manganese rather than a specific oxide of manganese.

Total manganese exposure also becomes an issue because manganese is an essential element and oral exposures occur. It would be desirable to know the effective target site doses and apportion the dose to both the inhalation and oral routes of exposure. However, given the lack of data regarding both oral and inhalation pharmacokinetics under environmental conditions, such quantitative apportionment is not possible.

In any case, the absorbed doses estimated above are not identical to the target site doses, although there is an unmeasured relationship. Until the data specified above become available, we are left with the conclusion that certain inhalation exposures, such as those in the occupational studies, cause adverse effects, and the NOAEL for humans has not been experimentally defined. Views could differ on what constitutes a NOAEL and a subthreshold level which would consider the range of susceptibilities in the general populace and uncertainty factors have been applied to account for extrapolation issues. By definition, the RfC is not intended to be an absolutely accurate value, as it encompasses an order of magnitude uncertainty. However, it is based on inhalation data. To take an oral NOAEL level and extrapolate to inhalation levels, without knowing the quantitative pharmacokinetic relationships between oral and inhalation exposures, has less scientific validity.

Chronic manganese poisoning in workers has been recognized since 1837.

The primary effects associated with manganese toxicity from inhalation exposure in humans are signs and symptoms of CNS toxicity (manganism) and pneumonia. Manganism is believed to result from disturbances in the extrapyramidal motor system. Canavan et al. (1934) reported the occurrence of diffuse cellular changes in the cerebral cortex and cerebellum, degeneration of nerve cells, satellitosis, and gliosis in the basal ganglia in a manganese

miner. The observed CNS toxicity can be divided into two stages: the first is dominated by psychological disturbances that subside if manganese exposure is

terminated; the second is predominantly a neurological disturbance, which occurs with continued manganese exposure and is not reversible. Manganese

neurotoxicity can involve psychiatric as well as neurobehavioral disturbances. In some cases these effects may be reversible; in others, the effects may persist even after termination of manganese exposure. Headache and somnolence followed by insomnia and fatigability are some of the earlier observed symptoms. If exposure is continued, speech and gait disturbances, tremor,

mask-like face, postural instability, emotional instability and hallucinations may occur. Numerous investigators have reported CNS effects in workers exposed to manganese dust or fumes (Badawy and Shakour, 1984; Chandra et al.,

1981; Cook et al., 1974; Emara et al., 1971; Flinn et al., 1941; Iregren,

1990; Rodier, 1955; Roels et al., 1987; Schuler et al., 1957; Smyth et al.,

1973; Tanaka and Lieben, 1969). Although there is an extensive database on

CNS effects in workers, limitations in the studies preclude describing a quantitative dose/response relationship. Manganese concentrations are often

presented as a broad range and particle size distribution and/or chemical characterization is not reported or adequately characterized. In addition,

the occurrence of other chemicals at the factory is often not reported. Despite the limitations of these studies, they do provide information for identifying an effect level; psychological disturbances and/or neurological

disturbances appear to be associated with long-term exposure to levels of manganese exceeding 0.25 mg/cu.m (Badawy and Shakour, 1984; Chandra et al.,

1981; Roels et al., 1987).

Lauwerys et al. (1985) reported the results of a fertility questionnaire administered to male factory workers (n=85) exposed to manganese dust. This

was the same population of workers for which Roels et al. (1987) reported increased prevalence of respiratory symptoms and psychomotor disturbances.

The range of manganese levels in the breathing zone was 0.07-8.61 mg/cu.m.,

with a median concentration of 0.97 mg/cu.m. The particle size distribution,

as well as the presence of other compounds, was not reported. Average length

of exposure was 7.9 years (range of 1-19 years). A group of workers (n=81)

with a similar workload was used as a control group. The number of births

expected during different age intervals of the workers (16-25, 26-35, 36-45) was calculated on the basis of the reproductive experience of the control employees during the same period. A decrease in the number of children born to workers exposed to manganese dust during the ages of 16-25 and 26-35 was observed. No difference in the sex ratio of the children was observed. The same apparent LOAEL(HEC) as was identified in Roels et al. (1987) for psychomotor and respiratory effects is identified in this study for human reproductive effects (0.34 mg/cu.m).

Workers exposed to manganese dust have a higher incidence of respiratory effects. An increased incidence of colds, bronchitis and pneumonia was reported in workers exposed to manganese dust (Lloyd-Davies, 1946; Lloyd-Davies and Harding, 1949; Roels et al., 1987) and junior high school students living near a ferromanganese factory (Nogawa et al., 1973). As discussed in regard to the CNS toxicity, the study limitations preclude the establishment of a dose-response relationship. Similar respiratory effects were also observed in animals (Lloyd-Davies, 1946; Lloyd-Davies and Harding, 1949; Shiotsuka, 1984; Suzuki et al., 1978). Other effects observed in humans include hematological (Chandra et al., 1981; Flinn et al., 1941; Kesic and Hausler, 1954), cardiovascular (Saric and Hrustic) and reproductive effects (Cook et al., 1974; Emara et al., 1971; Lauwerys et al., 1985; Rodier, 1955).

Workers employed in three different factories (30-35 workers/factory) and 30 matched controls were examined for neurological and psychological alterations (Badawy and Shakour, 1984). The mean concentrations of atmospheric manganese for the three plants were 1.0, 3.0, and 7.0 mg/cu.m. The specific manganese compound and other contaminants were not reported. An increased incidence of headache, involuntary movements, fatigue and exhaustion, sleep disturbances, sialorrhea, seborrhea, speech disturbances, gait disturbance, exaggerated reflexes, depression, hallucination, and prolonged reaction time were observed in workers exposed to manganese. The most common effects were headache, involuntary movements, fatigue, and exhaustion. The incidence of headaches; involuntary movements; disturbances in sleep, speech, and gait; and exaggerated reflexes were significantly increased with increasing duration of employment. Significant effects were

observed in all three plants, thereby indicating the a LOAEL of 1.0 mg/cu.m in this study. Concentration-response relationships for the incidence of involuntary movements, speech disturbances, gait disturbances and hallucinations were observed. No correlation between air and blood manganese levels was observed. From these data a LOAEL(HEC) of 0.36 mg/cu.m was calculated.

Nogawa et al. (1973) examined the possibility that high atmospheric manganese levels would result in respiratory effects in junior high school students. A questionnaire concerning subjective abnormalities in the eyes and throat was distributed to students attending junior high schools that were 100 m (enrollment=1258) and 7 km (enrollment size=648) away from a plant that primarily produced ferromanganese. The authors did not note socioeconomic variables were controlled. The atmospheric manganese level 100 m from the plant was reported to be 0.004 mg/cu.m. Levels of manganese in the school were not measured. In addition, the authors did not attempt to quantify the amount of manganese the children were exposed to when they were not in school. Other heavy metals, including cadmium, were present, but only manganese and iron levels were high compared to other cities. Over 98% of the students completed the questionnaires. Among the male students in the school 100 m away from the plant, a significant increase in the number of students reporting each of the following symptoms was observed: sputum always in the winter on arising, clogged nose, nose colds frequently in the summer, throat symptoms (swelling and soreness), past history of sinus empyema and an increase in the number of students with family members having coughs and sputum lasting for longer than 2 months. In the female students 100 m from the plant, the incidence of clogged nose, nose colds and throat symptoms in the winter was increased. When the male and female students were combined, a statistically significant increase in the incidence of eye symptoms and past history of pneumonia was observed. Among students enrolled in the school located 100 m from the plant and living closest to the plant, more students reported throat swelling and soreness in the summer and past history of pneumonia than did other students. The pulmonary lung function tests revealed statistically significant decreases in vital capacity, forced expiratory vital capacity at 1 second, and the 1-second ratio in the students attending the

school closest to the plant. Of the students living <500 m, 500-1000 m or 1000-1500 m away from the ferromanganese plant for >3 years, the lowest 1-second ratio was observed in the students living <500 m away. Distance of residency from the plant did not influence the 1-second ratios for students living at the place of residency for <3 years. The 1-second ratios in the students living <500 m away for <3 years was not different from the control students. These data suggest that a concentration-duration of exposure relationship may exist between manganese and pulmonary effects. The LOAEL(HEC) calculated from this study is  $4E-3$  mg/cu.m.

An increased incidence of pneumonia was observed in men employed at a potassium permanganate manufacturing facility during an 8-year period (n=40-124) as compared with a control group of workers (n>5000) (Lloyd-Davies, 1946). The levels of manganese in the dust ranged from 0.7-38.3 mg/cu.m of which 43-54%, respectively, was manganese dioxide (0.3-21 mg MnO<sub>2</sub>/cu.m, 0.2-13.2 mg Mn/cu.m). Approximately 80% of the particles were <0.2  $\mu$ m and nearly all were <1  $\mu$ m. The other major compounds in the dust included calcium and potassium; barium (1%) and sodium (0.1%) were also detected in the dust. The levels of calcium and potassium in the dust were not reported. Trace amounts of silica, iron and lithium were also detected. The incidence of pneumonia in the workers was 26 per 1000, compared to an average of 0.73 per 1000 in the control group. All cases were diagnosed as lobar or bronchopneumonia. Workers also complained of bronchitis and nasal irritation. In a continuation of the Lloyd-Davies (1946) study, Lloyd-Davies and Harding (1949) reported the results of sputum and nasopharynx cultures for four men diagnosed as having lobar or bronchopneumonia. With the exception of one of these cases, Lloyd-Davies and Harding (1949) concluded that it was unlikely that bacterial infection played a primary role in producing the consolidation present in the lung and that manganese dust, without the presence of other factors, caused the observed pneumonitis. Based upon the range of exposure to manganese (0.2-13.2 mg/cu.m), a LOAEL(HEC) range of 0.07-4.7 mg/cu.m can be estimated.

Saric et al. (1977) examined 369 workers in a ferroalloy plant. Workers in two other plants (electrode plant, n=190; aluminum rolling mill, n=204)

served as controls. The ferroalloy plant workers were exposed to 0.3-20.41 mg/cu.m manganese; the manganese levels in the electrode plant and aluminum rolling mill were 0.002-0.03 mg/cu.m and 0.00005-0.00007 mg/cu.m, respectively. The workers were exposed to either manganese dust or fumes. The manganese compound or compounds that the workers were exposed to was not reported. A significant increase in the following subjective symptoms was observed in the ferroalloy plant workers: fatigue, bad mood, irritability and hand tremor. One or more sign(s) of neurological impairment (e.g., tremor, pathological reflexes) was observed in 16.8 and 5.8% of the workers in the ferroalloy plant and electrode plant, respectively. A significant decrease in systolic blood pressure without a change in diastolic blood pressure was also reported in the ferroalloy plant workers (Saric and Hrustic, 1975). Saric and Lucic-Palaic (1977) reported that in these groups of workers, manganese exposure and smoking might have a possible synergistic effect on the occurrence of respiratory symptoms.

Chronic manganese psychosis (16.6%), neuropsychiatric manifestations (22.2%), hemi-parkinsonism (2.7%) and choreoathetosis (2.7%) were observed in 36 workers employed in the dry battery industry (Emara et al., 1971). The workers were exposed to a dust containing 65-70% manganese dioxide (6.8-42.4 mg/cu.m). Contaminants in the dust included ammonium chloride, zinc oxide, graphite, acetylene black, ammonium hydroxide, cerium thorium nitrate, magnesium nitrate, and mercuric chloride. The particle size distribution was not reported. The psychological manifestations included headache, memory disturbances, sleep disturbances, uncontrollable laughter, sexual impotence or diminished libido, impulsive acts, uncontrollable weeping, irritability or depression, and hallucinations.

Smyth et al. (1973) observed 71 workers exposed to manganese dust or fumes and 71 matched controls. The manganese levels in the fumes (primarily as manganese tetroxide) were 0.12-13.3 mg/cu.m and the majority of the particles were <2 microns in size. The manganese dust was mainly ferromanganese, with small amounts of manganosite (MnO), hausmannite (manganese tetroxide), and

iron oxide. The manganese level in the dust ranged from 2.1-12.9 mg/cu.m.; 95% of the particles were <5 microns in size. Neurological examination of the workers revealed five workers with signs of CNS impairment. Three of these workers were exposed to manganese fumes and the other two to manganese dust. The five affected were exposed to the upper end of the exposure range. It is unclear if other workers exhibited signs of neurobehavioral problems.

The available evidence obtained from small laboratory animals indicates that rats may display some of the neurochemical changes associated with manganism in humans; however, they do not exhibit the wide range of behavioral manifestations described in primates (U.S. EPA, 1984). Manganese accumulation appears to be relatively high in pigmented substantia nigra tissues. Since the primate (but not rodent substantia nigra) shows pigmentation, there is some basis for assuming species differences in accumulation and toxicity of manganese.

Because the deposition and retention in the respiratory tract is dependent on particle size, the particle size distribution of the atmospheric manganese is likely to play a role in respiratory tract damage. Particle size of the manganese dust was often not reported in the occupational studies; therefore, comparisons between human and experimental animal data are difficult. However, the experimental animal data support the findings in manganese workers that manganese exposure results in an increased incidence of pneumonia (Shiotsuka, 1984), pulmonary congestion (Nishiyama et al., 1975), and pulmonary emphysema (Suzuki et al., 1978).

Groups of four female Rhesus monkeys were exposed to 0 or 30 mg/cu.m manganese dust (particle size was <5 micron) for 6 hours/day, 5 days/week for 2 years. Regular observations did not reveal any behavioral or abnormal neurological signs. However, the monkeys were not tested for neurobehavioral dysfunction as part of the protocol and the report of lack of symptoms is based on cage side observation. Decreased dopamine levels were measured in the caudate and globus pallidus. The respiratory tract was not examined (Bird et al., 1984).

Male and female Sprague-Dawley rats (15/sex/group) and Squirrel monkeys (4/sex/group) were continuously exposed to 0, 0.012, 0.113 or 1.15 mg Mn/cu.m

manganese tetroxide for 9 months. The equivalent aerodynamic diameter of the particles was 0.11 microns. The atmosphere generation system used was designed to simulate the manganese tetroxide levels produced by an internal combustion engine burning gasoline containing methylcyclopentadienyl manganese tricarbonyl (Ulrich et al., 1979a). A statistically significant increase in hemoglobin and mean corpuscular hemoglobin concentration was observed at 1.15 mg/cu.m in both species. No histopathological alterations were observed in the lungs, larynx, pharynx, trachea, adrenals, or kidneys (Ulrich et al., 1979b). No consistent changes in lung function, electromyogram activity and limb tremor were observed in the monkeys (Ulrich et al., 1979c).

Groups of two to three rhesus monkeys were exposed to manganese dioxide dust at concentrations of 0, 0.7 or 3 mg Mn/cu.m for 22 hours/day, 7 days/week for 10 months. Hyperplasia of peribronchial tissue, pulmonary emphysema and atelectasis, exudate in bronchioles and thickening of the alveolar wall were observed in the exposed monkeys. The severity of the effects increased with concentration (Suzuki et al., 1978).

Groups of male Swiss mice (96/group) were exposed to 0 or 71.73 mg Mn/cu.m (TWA) manganese dioxide for 7 hours/day, 5 days/week for 16-32 weeks. The mice exposed to manganese executed more rearings in the open field and longer passive avoidance latencies (Morganti et al., 1985).

Male albino rats (74/group) and male golden hamsters (60/group) were exposed to automobile emissions for 56 consecutive days. The fuel used consisted of indolene "clear" to which methylcyclopentadienyl manganese tricarbonyl at 0.25 g Mn/gallon was added. During the 8-hour exposure, the animals were housed in either irradiated or nonirradiated chambers. For the rats, two of the exposed groups (irradiated and nonirradiated) were fed a low-manganese diet; a control group was also fed this diet. The other two rat groups consisted of a control group fed a typical laboratory diet and an exposed group in an irradiated chamber fed a typical diet. Manganese-exposed hamsters were fed a normal diet and housed in irradiated or nonirradiated chambers. The concentration of manganese in the irradiated chamber was 0.117 mg/cu.m (particle size = 0.29 micron); the level in the nonirradiated chambers was 0.131 mg/cu.m (particle size = 0.26 micron). At necropsy, no gross



abnormalities were noted except for "the usual chronic respiratory disease lesions in rats". No histopathological lesions attributable to the increased concentration of manganese were observed in the rats or hamsters except for thickening of the cuboidal epithelium in the terminal bronchiole in 21% of the irradiated exhaust treatment, 14% in the nonirradiated exhaust group, and 6% in the controls. Increased tissue manganese levels were observed in both species. The NOAEL(HEC) for rats is 0.12 mg/cu.m (Moore et al., 1975).

Groups of eight male rabbits (strain not specified) were exposed to 0, 1.1, or 3.9 mg Mn/cu.m manganese chloride (particle size 1 micron), 6 hours/day, 5 days/week for 4-6 weeks. No changes in lung morphology were observed (Camner et al., 1985).

Male and female Sprague-Dawley rats (3/sex/group) were exposed to 0, 68, 130, or 219 mg/cu.m manganese dioxide (0, 43, 82 or 138 mg Mn/cu.m), 6 hours/day, 5 days/week for 2 weeks. The MMAD was 3.17 microns, with a sigma g of 2.92. No hematological alterations (hemoglobin, hematocrit, red and white blood cells and mean corpuscular volume) were observed. A concentration-related increase in the incidence of pneumonitis and an increase in wet lung weight were observed. The severity of the pneumonitis increased with concentration. At 43 mg/cu.m, focal pneumonitis with interstitial hypercellularity was observed. Diffuse pneumonitis and mature granulomas were observed in the rats exposed to 138 mg/cu.m manganese. Based upon the pulmonary effects, a LOAEL(HEC) of 7.2 mg/cu.m was calculated (Shiotsuka, 1984).

Nishiyama et al. (1975), as summarized in U.S. EPA (1984), observed pulmonary congestion in monkeys exposed to 0.7 or 3.0 mg/cu.m manganese dioxide, 22 hours/day for 5 months. The pulmonary changes in the monkeys exposed at 0.7 mg/cu.m occurred later and were less severe compared to the 3.0 mg/cu.m group. Groups of mice were exposed to manganese dioxide at 0.7 or 3.0 mg/cu.m for 22 hours/day for 2 weeks. Reversible inflammatory changes were observed in both groups. Two months after termination of exposure, the inflammation was not present and desquamation of the bronchial epithelium was observed.

One of the primary effects of manganese exposure in humans is an increased prevalence of respiratory symptoms (pneumonia, bronchitis, colds, and coughs) (Lloyd-Davies, 1946; Nogawa et al., 1973; Roels et al., 1987). Respiratory effects have also been reported in animals (Nishiyama et al., 1975; Shiotsuka,

1984; Suzuki et al., 1978). It is unlikely that exposure to manganese is solely responsible for the increased prevalence of respiratory symptoms. Rather, manganese exposure probably increases susceptibility to infection.

This is supported by several animal studies that have demonstrated immunotoxicity following exposure to manganese and *Streptococci*, *Enterobacter* or *Klebsiella* (Adkins et al., 1980; Bergstrom, 1977; Graham et al., 1980; Lloyd-Davies, 1946; Maigetter et al., 1976).

Male and female guinea pigs (sample size not reported) were exposed to 22 mg/cu.m manganese dioxide (13.9 mg Mn/cu.m) for 24 hours; 87% of the particles were <3 microns in size. Groups of guinea pigs were exposed to *Enterobacter* cloacae 1 day prior to manganese exposure, immediately before manganese exposure, or immediately after manganese exposure. The decrease in the clearance of manganese dioxide from the lungs, decrease in lung macrophages, and increase in the number of lung leukocytes observed in animals exposed to *Enterobacter* 1 day prior to manganese exposure were significant when compared to the manganese exposure-only group (Bergstrom, 1977).

CD-1 mice were exposed to various levels of manganese tetroxide for 2 hours. A concomitant control group was used. Following exposure to manganese, the mice were exposed to *Streptococcus pyogenes* aerosol for 20 minutes. A concentration-related increase in the difference in mortality between the manganese-exposed mice and the control mice was observed. Based on the regression line of this positive correlation (correlation coefficient of 0.71), Adkins et al. (1980) concluded that exposure to <0.62 mg/cu.m would result in a mortality rate that is <10% of the controls'. Other effects in the manganese-exposed animals (compared to the control mice) include: earlier occurrence of *Streptococcal* bacteremia and a consistent concentration-response relationship between the amount of manganese retained in the lungs, the enhanced mortality rate, reduced initial clearance and subsequent enhanced growth of the *Streptococci*. In addition, Adkins et al. (1980) observed that immunity against *Streptococci* did not counteract the toxic effects of manganese oxide inhalation and consequent *Streptococci* infection.

Groups of male CD-1 mice were exposed to 0 or 109 mg manganese dioxide/cu.m (68.8 mg Mn/cu.m) 3 hours/day for 1-4 days. One to 5 hours after the termination of manganese exposure, the mice were exposed to *Klebsiella pneumoniae* aerosol. Increased mortality and decreased survival time were

observed in the animals exposed to manganese for one 3-hour period and K. pneumoniae 5 hours later. When the period between the end of manganese exposure and administration of K. pneumoniae was extended from 1 to 5 hours,

there was a significant increase in mortality. Another group of mice was exposed to influenza A/PR/8/34 virus 24 or 48 hours prior to exposure to manganese dioxide. When the length of time between exposure to manganese and influenza virus was increased, there was a significant increase in mortality, decreased survival times, and increased pulmonary lesions (Maigetter et al., 1976).

Mice were exposed to the dust from a potassium permanganate manufacturing plant (Lloyd-Davies, 1946). The animals were exposed to the 70% manganese dioxide dust 2 times/day for 120 or 70 minutes for 15-21 days. Upon exposure termination, the animals were exposed to pneumococci and/or streptococcus hemolyticus. Of the 60 mice that were exposed for 120-minute sessions, nine died or were killed in extremis after 11 days. Several of the animals that were exposed for 70 minutes also died. Bronchopneumonia, swelling of the bronchial epithelium, and mononuclear infiltration were observed in these animals. No change in susceptibility to pneumococci was observed in the exposed mice (Lloyd-Davies, 1946).

Lloyd-Davies and Harding (1949) administered a single intratracheal injection of manganese dioxide (10 mg MnO<sub>2</sub>, equivalent to 6.3 mg Mn) or manganese chloride (5 or 50 mg MnCl<sub>2</sub>, equivalent to 2.18 or 21.8 mg Mn). The rats were sacrificed from 1 hour to 18 months (MnO<sub>2</sub>) or 8 days (MnCl<sub>2</sub>). Bronchiolar epithelium inflammation, widespread pneumonia, and granulomatous reactions were observed in the rats administered manganese dioxide. Pulmonary edema was observed in the group exposed to manganese chloride.

Female HA/ICR mice were exposed to 48.9 mg Mn /cu.m manganese dioxide 7 hours/day, 5 days/week for 4 months and were bred to unexposed males. The pregnant mice were exposed during gestational days 1-18. Decreased body weight and impaired neurobehavioral performance (open field, rotorod and exploration) were observed in the offspring. A decrease in rotorod performance was also observed in the offspring of non-exposed mice that were fostered to manganese-exposed females during lactation. Thus, balance and coordination were affected by either gestational or post-partum exposure to

manganese dioxide (Massaro et al., 1980).

#### I.B.5. CONFIDENCE IN THE INHALATION RfC

Study: Medium  
Data Base: Medium  
RfC: Medium

Confidence in the principal study (Roels et al., 1987) is medium. The LOAEL for respiratory and CNS effects was supported by several other human studies (Chandra et al., 1981; Iregren, 1990; Nogawa et al., 1973; Badawy and Shakour, 1984). An adequate number of manganese workers were matched to an adequate number of control workers. Several limitations of the study preclude assigning higher confidence to it. No monitoring data were available to characterize past manganese levels. This is especially important because the production level at the factory increased with time; workers, therefore, may have been exposed to lower levels of manganese. In addition, particle size distribution, manganese compounds and other compounds present in the factory were not reported. Confidence in the data base is medium. The Chandra et al. (1981) study did not characterize exposure to other metals found in welding rods or adequately describe particle size and examined relatively few exposed subjects. The primary toxicological effects of exposure to airborne manganese have been fairly well characterized. However, limitations of the human studies preclude the establishment of a dose-response relationship. A no-effect level has not been identified. In addition, the effects of manganese on development and reproduction have not been adequately studied. Reflecting medium confidence in the key study and medium confidence in the data base, confidence in the inhalation RfC is medium.

#### I.B.6. EPA DOCUMENTATION AND REVIEW OF THE INHALATION RfC

Source Document -- This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation -- U.S. EPA, 1984.

Agency Work Group Review: 08/23/90, 09/19/90

Verification Date: 09/19/90

I.B.7. EPA CONTACTS (INHALATION RfC)

Kenneth A. Poirier / ORD -- (513)569-7531 / FTS 684-7531

Michael J. Davis / ORD -- (919)541-4162 / FTS 629-4162

(CAR) Carcinogenicity Assessment:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

NOTE: Manganese is an element considered essential to human health.

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity

Basis -- Existing studies are inadequate to assess the carcinogenicity of manganese.

II.A.2. HUMAN CARCINOGENICITY DATA

None.

II.A.3. ANIMAL CARCINOGENICITY DATA - Inadequate

DiPaolo (1964) subcutaneously or intraperitoneally injected DBA/1 mice with 0.1 mL of an aqueous solution of manganese chloride twice weekly for 6 months. A larger percentage of the mice exposed subcutaneously (24/36; 67%) and intraperitoneally (16/39; 41%) to manganese developed lymphosarcomas compared with controls injected with water (16/66; 24%). In addition, tumors appeared earlier in the exposed groups than in the control groups. The incidence of tumors other than lymphosarcomas (i.e., mammary adenocarcinomas, leukemias, injection site tumors) did not differ significantly between the exposed groups and controls. A thorough evaluation of the results of this study was not possible because the results were published in abstract form.

Stoner et al. (1976) tested manganous sulfate in a mouse lung adenoma screening bioassay. Groups of strain A/Strong mice (10/sex), 6-8 weeks old, were exposed by intraperitoneal injection to 0, 6, 15 or 30 mg/kg manganous sulfate 3 times/week for 7 weeks (a total of 21 injections). The animals were

observed for an additional 22 weeks after the dosing period, before sacrifice at 30 weeks. Lung tumors were observed in 12/20, 7/20, and 7/20 animals in the high, medium, and low dosage groups, respectively. The percentage of mice with tumors was elevated, but not significantly, at the highest dose level (Fisher Exact test) compared with that observed in the vehicle controls. In addition, there was an apparent increase in the average number of pulmonary adenomas per mouse both at the mid and high doses, as compared with the vehicle controls (10 mice/sex), but the increase was significant only at the high dose (Student's t-test,  $p < 0.05$ ).

In the mouse lung adenoma bioassay, certain specific criteria should be met in order for a response to be considered positive (Shimkin and Stoner, 1975). Among these criteria are an increase in the mean number of tumors per mouse and an evident dose-response relationship. While the results of this study are suggestive of carcinogenicity, the data cannot be considered conclusive since the mean number of tumors per mouse was significantly increased at only one dose, and the evidence for a dose-response relationship was marginal.

Furst (1978) exposed groups of F344 rats (25/sex) intramuscularly or by gavage to manganese powder, manganese dioxide, and manganese (II) acetylacetonate (MAA). Treatment consisted of either 9 i.m. doses of 10 mg each of manganese powder or manganese dioxide, 24 doses of 10 mg manganese powder by gavage, or 6 i.m. doses of 50 mg of MAA. In addition, female swiss mice (25/group) were exposed intramuscularly to manganese powder (single 10 mg dose) and manganese dioxide (6 doses of 3 or 5 mg each). There was an increased incidence of fibrosarcomas at the injection site in male (40%) and female (24%) rats exposed intramuscularly to MAA compared with vehicle controls (4% male, 4% female). EPA (1984) determined that these increases were statistically significant and noted that the study results regarding MAA, an organic manganese compound, cannot necessarily be extrapolated to pure manganese or other inorganic manganese compounds. No difference in tumor incidence was found between rats and mice exposed to manganese powder and manganese dioxide and controls.

Sunderman et al. (1974, 1976) exposed male 344 rats to 0.5 to 4.4 mg manganese dust intramuscularly and found that no tumors were induced at the injection site. It was further observed that co-administration of manganese with nickel subsulfide resulted in decreased sarcoma production by comparison to nickel subsulfide alone. Subsequent studies by Sunderman et al. (1980) suggest that manganese dust may inhibit local sarcoma induction by benzo(a)pyrene.

Witschi et al. (1981) exposed female A/J mice intraperitoneally to 80 mg/kg methylcyclopentadienyl manganese tricarbonyl (MMT) and found that although cell proliferation was produced in the lungs, lung tumor incidence did not increase.

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

None.

#### II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

##### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1984. Health Assessment Document for Manganese. Office of Research and Development, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. EPA 600/8-83-013F.

U.S. EPA. 1988. Drinking Water Criteria Document for Manganese. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. ECAO-CIN-D008. (External Review Draft).

##### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The Drinking Water Criteria Document for Manganese has received OHEA review.

Agency Work Group Review: 05/25/88

Verification Date: 05/25/88

##### II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Cynthia Sonich-Mullin / ORD -- (513)569-7523 / FTS 684-7523

Julie Du / ODW -- (202)382-7583 / FTS 382-7583



Option? TYPE 12/2

File 12; Entry 1; Acca No. 1370

(CAS) CAS Registry Number: 7439-97-6

(MAT) Material Name: Mercury (Inorganic)

(SYN) Synonyms:  
hydragyrum;  
Mercury

(UPD) Update Date: 05-01-91

(EFF) Effective Date: 07-01-91

(STAT) Status:  
STATUS OF DATA FOR Mercury (Inorganic)

File On-Line 09-07-88

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	pending	
Inhalation RfC Assessment (I.B.)	pending	
Carcinogenicity Assessment (II.)	on-line	05-01-91
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	no data	
Supplementary Data (V.)	no data	

(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

A risk assessment for this substance/agent is under review by an EPA work group.

#### I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

A risk assessment for this substance/agent is under review by an EPA work group.

#### (CAR) Carcinogenicity Assessment:

##### II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

##### II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

##### II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity

Basis -- No human data are available. Animal and supporting data are inadequate.

##### II.A.2. HUMAN CARCINOGENICITY DATA

None.

##### II.A.3. ANIMAL CARCINOGENICITY DATA

When 39 BD III and BD IV rats were injected i.p. over 2 weeks with 0.1 ml metallic mercury and observed for their lifetimes, sarcomas were seen only in those tissues that had been in direct contact with the metal (Druckrey et al., 1957). No concurrent controls were reported.

##### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Mitsumori et al. (1981) fed groups of 60 male and 60 female SPF ICR mice 0, 15 or 30 ppm methyl mercury chloride in the diet for up to 78 weeks. The majority of the 30 ppm groups died from neurotoxicity by week 26. Histopathology on kidney tissue from all animals surviving after 53 weeks revealed renal tumors in 13/16 males in the 15 ppm group (2 adenomas, 11 adenocarcinomas). One adenoma was detected among 37 controls surviving to week 53 or beyond, and no tumors were seen in either control or exposed females. The possible presence of tumors at other sites was not reported in this preliminary communication.

Methyl mercury hydroxide administered in the diet to *Drosophila melanogaster* at 5 mg/L induced chromosomal nondisjunction. Methyl and phenyl mercury produced small increases in the rate of point mutations (Ramel, 1972).

The relevance of data from studies of organic mercury to the possible carcinogenicity of inorganic mercury is uncertain.

#### II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

##### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1987. Drinking Water Criteria Document for Mercury. Prepared for the Office of Drinking Water, Washington, DC. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. ECAO-CIN-025, February, 1987.

##### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The 1987 Drinking Water Criteria Document for Mercury has received Agency and external review.

Agency Work Group Review: 01/13/88

Verification Date: 01/13/88

##### II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

W. Bruce Peirano / ORD -- (513)569-7540 / FTS 684-7540

Krishan Khanna / ODW -- (202)382-7588 / FTS 382-7588

Option? CAS/108101

File: 7 Count: 1

Option? TYPE 7/2

File 7; Entry 1; Accession No. 1173

(CAS) CAS Registry Number: 108-10-1

(MAT) Material Name: Methyl isobutyl ketone (MIBK)

(SYN) Synonyms:

HEXON;  
HEXONE;  
ISOBUTYL-METHYLKETON;  
ISOBUTYL METHYL KETONE;  
ISOPROPYLACETONE;  
KETONE, ISOBUTYL METHYL;  
METHYL-ISOBUTYL-CETONE;  
METHYLISOBUTYLKETON;  
Methyl Isobutyl Ketone;  
4-METHYL-PENTAN-2-ON;  
2-METHYL-4-PENTANONE;  
4-METHYL-2-PENTANONE;  
METILISOBUTYLCHETONE;  
4-METILPENTAN-2-ONE;  
METYLOIZOBUTYLOKETON;  
MIBK;  
MIK;  
2-PENTANONE, 4-METHYL-;  
RCRA WASTE NUMBER U161;  
SHELL MIBK;  
UN 1245

(UPD) Update Date: 03-01-91

(EFF) Effective Date: 07-01-91

(STAT) Status:

STATUS OF DATA FOR MIBK

File On-Line 03-31-87

Category (section)	Status	Last Revised
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Oral RfD Assessment (I.A.)	withdrawn	03-01-91
Inhalation RfC Assessment (I.B.)	pending	
Carcinogenicity Assessment (II.)	no data	

Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	on-line	03-01-88
Supplementary Data (V.)	no data	

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(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

The Oral RfD for this substance has been withdrawn pending further review by the RfD/RfC Work Group.

Contact: Michael L. Dourson / ORD / FTS/684-7544 or 513/569-7544

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

A risk assessment for this substance/agent is under review by an EPA work group.

(CAR) Carcinogenicity Assessment:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

This substance/agent has not been evaluated by the U.S. EPA for evidence of human carcinogenic potential.

(REGS) Regulations:

III. HEALTH HAZARD ASSESSMENTS FOR VARIED EXPOSURE DURATIONS

IV. U.S. EPA REGULATORY ACTIONS

IV.A. CLEAN AIR ACT (CAA)

No data available

IV.F. RESOURCE CONSERVATION AND RECOVERY ACT (RCRA)

IV.F.1. RCRA APPENDIX IX, for Ground Water Monitoring

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

IV.G. SUPERFUND (CERCLA)

IV.G.1. REPORTABLE QUANTITY (RQ) for Release into the Environment

Value (status) -- 5000 pounds (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- The final adjusted RQ for this substance is 5000 pounds, based on application of the secondary criterion of biodegradation to the primary criterion RQ of 1000 pounds determined by ignitability. Available data indicate a flash point of 64F and a boiling point of 224F. The final RQ takes into account the biodegradation of methyl isobutyl ketone [BOD5 - 4.4%, BOD5 - 56% (sewage seed), BOD20 - 57%, BOD20 - 65%]. Therefore, the 1000-pound RQ based on ignitability has been adjusted upward one level to 5000 pounds.

Reference -- 50 FR 13456 (04/04/85)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

File 4; Entry 1; Accession No. 1436

(CAS) CAS Registry Number: 91-20-3

(MAT) Material Name: Naphthalene

(SYN) Synonyms:

Naphthalene;  
Albocarbon;  
Caswell No. 587;  
Dezodorator;  
EPA Pesticide Chemical Code 055801;  
HSDB 184;  
MOTH BALLS;  
MOTH FLAKES;  
Naftalen [Polish];  
Naftaleno [Spanish];  
Naphtalene [French];  
Naphthalene;  
Naphthalin;  
Naphthaline;  
Naphthene;  
NAPTHALENE, molten;  
NCI-C52904;  
NSC 37565;  
RCRA WASTE NUMBER U165;  
TAR CAMPHOR;  
UN 1334;  
UN 2304;  
WHITE TAR

(UPD) Update Date: 12-01-90

(EFF) Effective Date: 10-01-91

(STAT) Status:

STATUS OF DATA FOR Naphthalene

File On-Line 12-01-90

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	pending	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	12-01-90
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	no data	

Supplementary Data (V.)

no data

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(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

A risk assessment for this substance/agent is under review by an EPA work group.

(CAR) Carcinogenicity Assessment:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity

Basis -- Based on no human data and inadequate data from animal bioassays.

II.A.2. HUMAN CARCINOGENICITY DATA

None.

II.A.3. ANIMAL CARCINOGENICITY DATA

Inadequate. The National Toxicology Program is currently evaluating naphthalene for carcinogenicity in mice by the inhalation route; final results are not yet available.

A group of 28 rats (in-house strains BDI and BDIII) was exposed to a diet supplemented with naphthalene, 6 times/week (Schmahl, 1955). Treatment was stopped when total dose was 10 g/rat. The average daily dose was approximately 10 to 20 mg/day (approximately 30 to 60 mg/kg/day). Tumors were evaluated in animals that died spontaneously at about 700 to 800 days of age. No carcinogenic responses were reported.

In a short-term pulmonary tumor bioassay, Adkins et al. (1986) exposed groups of 30 female A/J strain mice by inhalation to 0, 10, or 30 ppm naphthalene for 6 hours/day, 5 days/week for 6 months. While naphthalene caused a statistically significant increase in the number of adenomas per mouse lung, there was no apparent dose-response. This assay is considered to be a short-term, in vivo, lung tumor assay.



Tsuda et al. (1980) administered a single gavage dose of 100 mg/kg naphthalene in corn oil to a group of 10 F344 rats (sex not specified) at 12 hours after partial hepatectomy. A vehicle control group of 10 rats was included. At 2 weeks after surgery, 2-acetylaminofluorene was added to the diet at 200 ppm to inhibit proliferation of "nonresistant" hepatocytes. After 1 week of dietary 2-acetylaminofluorene, a single 2.0 mL/kg dose of carbon tetrachloride was given to necrotize "nonresistant" hepatocytes and permit proliferation of "resistant" hepatocytes. Feeding of 2-acetylaminofluorene continued for 1 week, followed by a basal diet for 1 week. The rats were then sacrificed and livers were sectioned and histochemically examined for the number and size of gamma-glutamyl transpeptidase (GGT) positive foci. These foci contain cells that are "resistant" to the necrotizing effects of carbon tetrachloride and to the proliferation-inhibiting effects of 2-acetylaminofluorene and are considered to represent an early stage in the process of neoplastic transformation. Neither the number nor the size of GGT foci appeared to be increased in naphthalene-treated rats compared with vehicle controls.

A group of 10 rats (in-house strains BDI and BDIII) received intraperitoneal injections of naphthalene (20 mg/rat) once a week for 40 weeks (Schmahl, 1955). Another group of 10 rats served as a control group. Animals were evaluated after spontaneous death. No carcinogenic responses were reported.

Coal tar-derived naphthalene that contained approximately 10% unidentified impurities was administered to 40 white rats (sex unspecified) by seven subcutaneous injections of 500 mg/kg naphthalene in sesame oil at 2-week intervals. Lymphosarcomas were found in 5/34 surviving rats at 18 months (14.7%), whereas vehicle controls had a 2% incidence of these tumors. This study is of limited value because of the presence of potentially carcinogenic impurities in the naphthalene and because prior to injection carbofuchsin was applied dermally to the injection site (Knake, 1956).

Inbred black mice (25/group) were painted with 0.5% coal tar-derived naphthalene (10% unidentified impurities) in benzene 5 days/week for life.

Four treated mice developed leukemias in contrast to 0/21 vehicle controls; the

untreated control incidence was 0.4%. The value of this study for assessing carcinogenicity is very limited due to the presence of potentially carcinogenic impurities. Moreover, the vehicle in the study has been shown to cause leukemias (Knake, 1956). Other mouse skin-painting tests of naphthalene as a complete carcinogen and as an initiator of carcinogenicity were negative or inconclusive (Kennaway, 1930; Schmeltz et al., 1978).

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

With one exception naphthalene was not positive when tested in a variety of genotoxicity assays. In reverse mutation assays using *Salmonella typhimurium* strains TA97, TA98, TA100, TA1535, TA1537, TA1538, UTH8413 and UTH8414, naphthalene at concentrations of up to 2.5 mg/plate was not positive either with or without hepatic homogenates (McCann et al., 1975; Anderson and Styles, 1978; Florin et al., 1980; Gatehouse, 1980; Connor et al., 1985; Ho et al., 1981; Sakai et al., 1985; Mortelmans et al., 1986; Bos et al., 1988).

Narbonne et al. (1987) reported that in the presence of hepatic homogenates naphthalene at 5 and 10 ug/plate was mutagenic for *S. typhimurium* TA1538; however, naphthalene was not positive at concentrations of 50, 100 and 1000 ug/plate. There was no increase in forward mutation frequency for *Salmonella*.

At concentrations of up to 1.6 mM, naphthalene was not positive in *S. typhimurium* forward mutation assays (Kaden et al. 1979; Seixas et al., 1982).

In a DNA damage assay using *S. typhimurium* TA1535 Nakamura et al. (1987) reported that naphthalene at concentrations of up to 83 ug/mL was not positive. In phage induction assays using *Escherichia coli* as a host, naphthalene at concentrations of up to 2 mg/mL did not yield positive results

(Ho and Ho, 1981; Mamber et al. 1984). DNA damage assays with naphthalene were not positive in *E. coli* (Mamber et al., 1983) or in primary rat hepatocyte cultures (Sina et al., 1983). Transformation assays in Swiss mouse embryo cells (Rhim et al., 1974) and in rat embryo cells (Freeman et al., 1973) were not positive.

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

None.

II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

None.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

II.D.1. EPA DOCUMENTATION

U.S. EPA. 1986. Health and Environmental Effects Profile for Naphthalene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC. Final Draft. ECAO-CIN-P192, August, 1986.

U.S. EPA. 1990. Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons (PAHs). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. Final Draft. ECAO-CIN-D010, September, 1990.

II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The 1990 Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons has undergone Agency and external review.

Agency Work Group Review: 02/07/90

Verification Date: 02/07/90

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Rita S. Schoeny / ORD -- (513)569-7544 / FTS 684-7544

Robert E. McGaughy / ORD -- (202)260-5889 / FTS 260-5889

Option? TYPE 13/2

File 13; Entry 1; Accession No. 1271

(CAS) CAS Registry Number: 7440-02-0

(MAT) Material Name: Nickel, soluble salts

(SYN) Synonyms:

C.I. 77775;

NICHEL;

Nickel;

Nickel, soluble salts

(UPD) Update Date: 06-01-90

(EFF) Effective Date: 07-01-91

(STAT) Status:

STATUS OF DATA FOR Nickel, soluble salts

File On-Line 09-30-87

Category (section)	Status	Last Revised
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Oral RfD Assessment (I.A.)	on-line	06-01-90
Inhalation RfC Assessment (I.B.)	pending	
Carcinogenicity Assessment (II.)	message	
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	on-line	06-01-90
Supplementary Data (V.)	on-line	09-30-87

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(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

NOTE: The Oral RfD for nickel (soluble salts) may change in the near future

pending the outcome of a further review now being conducted by the Oral RfD Work Group.

#### I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
-----	-----	-----	---	-----
Decreased body and organ weights	NOAEL: 100 ppm diet (5 mg/kg/day)	100	3	2E-2 mg/kg/day
Chronic Rat Feeding Study	LOAEL: 1000 ppm diet (50 mg/kg/day)			
Ambrose et al., 1976				
-----				

\*Conversion Factors: 1 ppm = 0.05 mg/kg/day (assumed rat food consumption)

#### I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Ambrose, A.M., D.S. Larson, J.R. Borzelleca and G.R. Hennigar, Jr. 1976. Long-term toxicologic assessment of nickel in rats and dogs. J. Food Sci. Technol. 13: 181-187.

Ambrose et al. (1976) reported the results of a 2-year feeding study using rats given nickel sulfate hexahydrate in concentrations of 0, 100, 1000 or 2500 ppm as nickel (Ni) (estimated as 0, 5, 50, and 125 mg Ni/kg bw) in the diet. Body weights in the high-dose male and female rats were significantly decreased compared with controls. Body weight was also reduced at 1000 ppm; this reduction was significant for females at week 6 and from week 26 through 104, whereas males showed body weight reductions only at 52 weeks. Groups of female rats on the 1000 or 2500 ppm nickel diets (50 and 125 mg Ni/kg bw) had significantly higher heart-to-body weight ratios and lower liver-to-body weight ratios than controls. No significant effects were reported at 100 ppm (5 mg Ni/kg bw). The dose of 1000 ppm (50 mg Ni/kg bw) represents a LOAEL for this study, while the 100 ppm (5 mg Ni/kg bw) dose is a NOAEL. In this study, 2-year survival was poor, particularly in control rats of both sexes (death:

44/50); this raised some concern about the interpretation of the results of this study.

A subchronic study conducted by American Biogenics Corp. (U.S. EPA, 1986) also found 5 mg/kg/day to be a NOAEL, which supports the Ambrose et al. (1976) chronic NOAEL of 5 mg/kg/day. U.S. EPA (1986) reported that the 90-day study with nickel chloride in water (0, 5, 35, and 100 mg/kg/day) was administered by gavage to both male and female CD rats (30 animals/sex/group). The data generated in this study included clinical pathology, ophthalmologic evaluations, serum biochemistry, body and organ weight changes, and histopathologic evaluations of selected organs (heart, kidney, liver). The body weight and food consumption values were consistently lower than controls for the 35 and 100 mg/kg/day dosed males. Female rats in both high-dose groups had lower body weights than controls, but food consumption was unaffected by the chemical. Clinical signs of toxicity, such as lethargy, ataxia, irregular breathing, cool body temperature, salivation, and discolored extremities, were seen primarily in the 100 mg/kg/day group; these signs were less severe in the 35 mg/kg/day group. The 5 mg/kg/day group did not show any significant clinical signs of toxicity. There was 100% mortality in the high-dose group; 6/30 males and 8/30 females died in the mid-dose group (35 mg/kg/day). Histopathologic evaluation indicated that the deaths of 3/6 males and 5/8 females in the mid-dose group were due to gavage errors. At sacrifice, kidney, liver, and spleen weights for males treated at the 35 mg/kg/day dose level and right kidney weights for females treated at the 35 mg/kg/day dose level were significantly lower than controls. Based on the results obtained in this study, the 5 mg/kg/day nickel dose was a NOAEL, whereas the 35 mg/kg/day was a LOAEL for decreased body and organ weights.

#### I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RFD)

UF = 100. An uncertainty factor of 100 is used: 10 for interspecies extrapolation and 10 to protect sensitive populations. The nickel dietary study by Ambrose et al. (1976) identifying a NOAEL of 100 ppm (5 mg/kg/day) is supported by the subchronic gavage study in water (U.S. EPA, 1986), which indicated the same NOAEL (5 mg/kg/day). The uncertainty factor of 100 is therefore appropriate, since two studies support the NOAEL of 5 mg/kg/day.

MF - 3. A modifying factor of 3 is used because of inadequacies in the reproductive studies (RTI, 1987; Ambrose et al., 1976, see Additional Comments section). During the gestation and postnatal development of Flb litters in the RTI (1987) study, temperatures were about 10F higher than normal at certain times, which makes evaluation of this part of the reproductive study impossible. In the Ambrose et al. (1976) study there were some statistical design limitations, such as small sample size and use of pups rather than litters as the unit for comparison.

#### I.A.4. ADDITIONAL COMMENTS (ORAL RFD)

Ambrose et al. (1976) also reported reproductive toxicity of nickel, but the study had some statistical design limitations, such as small sample size and use of pups rather than litters as the unit for comparison. Furthermore, the results were equivocal and did not clearly define a NOAEL or LOAEL. The fact that nickel was administered in a laboratory chow diet containing milk powder, rather than in drinking water, in this study caused problems in quantification of nickel exposure when applying these data to drinking water situations.

In a 2-generation study (RTI, 1987), nickel chloride was administered in drinking water to male and female CD rats (30/sex/group) at dose levels of 0, 50, 250, and 500 ppm (0, 7.3, 30.8, and 51.6 mg/kg/day, estimated) for 90 days prior to breeding (10 rats/sex/group comprised a satellite subchronic nonbreeder group). At the 500 ppm dose level there was a significant decrease in the P-zero maternal body weights, along with absolute and relative liver weights. Thus, 250 ppm (30.8 mg/kg/day) was a NOAEL for P-zero breeders. Histopathology was performed for liver, kidney, lungs, heart, pituitary, adrenals, and reproductive organs to make this assessment. This NOAEL is higher than the NOAEL derived from the chronic Ambrose et al. (1976) and subchronic gavage (U.S. EPA, 1986) assays.

The number of live pups/litter was significantly decreased, pup mortality was significantly increased, and average pup body weight was significantly decreased in comparison with controls for the Fla generation (postnatal days 1-4) at the 500 ppm dose level (RTI, 1987). Similar effects were seen with Flb litters of P-zero dams exposed to 500 ppm nickel. In the 50 and 250 ppm

dose group, increased pup mortality and decreased live litter size were observed in the Flb litters. However, these effects seen with Flb litters are questionable because the room temperature tended to be 10F higher than normal at certain times (gestation-postnatal days) along with much lower levels of humidity. As evidenced in the literature, temperatures that are 10F above normal during fetal development cause adverse effects (Edwards, 1986). Therefore, the above results seen at the 50 and 250 dose levels cannot be considered as genuine adverse effects.

Flb males and females of the RTI (1987) study were randomly mated on postnatal day 70 and their offspring (F2a and F2b) were evaluated through postnatal day 21. This phase included teratologic evaluations of F2b fetuses. Evaluation of the data indicated that the 500 ppm dose caused significant body weight depression of both mothers and pups, and increased neonatal mortality during the postnatal development period. The intermediate dose, 250 ppm nickel, produced transient depression of maternal weight gain and water intake during gestation of the F2b litters. The 50 ppm nickel exposure caused a significant increase in short ribs (11%). However, since this effect was not seen in both of the higher dose groups, the reported incidence of short ribs in the 50 ppm group is not considered to be of biological significance.

Schroeder and Mitchener (1971) conducted a 3-generation study in which five mating pairs of rats were provided drinking water containing 5 mg Ni/L (estimated as 0.43 mg/kg bw). Results of this study indicated significant increases in neonatal mortality and number of runts born to exposed rats compared with controls. The major weakness of this study, however, is that the end result is based on a total of five matings. The matings were not randomized and the males were not rotated. The Schroeder and Mitchener (1971) study was conducted in an environmentally controlled facility where rats had access to food and water containing minimal levels of essential trace metals. Because of the interaction of nickel with other trace metals, the restricted exposure to trace metals (chromium was estimated as inadequate) may have contributed to the toxicity of nickel.

#### I.A.5. CONFIDENCE IN THE ORAL RfD

Study: Low  
Data Base: Medium  
RfD: Medium



The chronic study (Ambrose et al., 1976) was properly designed and provided adequate toxicologic endpoints; however, there was high mortality in the controls (44/50). Therefore, a low confidence is recommended for the study. The data base provided adequate supporting subchronic studies, one by gavage and the other in drinking water [P-zero animals of the RTI (1986) subchronic study]. A medium confidence level in the data base is recommended because there are inadequacies in the remaining reproductive study data. The RfD is adequately supported by the oral subchronic and reproductive studies, and until additional reproductive studies are available a medium confidence in the RfD is recommended.

#### I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

U.S. EPA. 1983. Health Assessment Document for Nickel. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. External Review Draft.

U.S. EPA. 1985. Drinking Water Criteria Document for Nickel - Quantification of Toxicological Effects Chapter Only. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. EPA 600/x-84-193-1.

Extensive Agency-wide Review, 1987

Agency RfD Work Group Review: 04/16/87, 05/20/87, 07/16/87

Verification Date: 07/16/87

#### I.A.7. EPA CONTACTS (ORAL RfD)

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Christopher T. DeRosa / ORD -- (513)569-7534 / FTS 684-7534

#### I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

A risk assessment for this substance/agent is under review by an EPA work group.

(CAR) Carcinogenicity Assessment:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

The U.S. EPA has not evaluated soluble salts of nickel, as a class of compounds, for potential human carcinogenicity. However, nickel refinery dust and specific nickel compounds - nickel carbonyl and nickel subsulfide - have been evaluated. Summaries of these evaluations are on IRIS.

(REGS) Regulations:

III. HEALTH HAZARD ASSESSMENTS FOR VARIED EXPOSURE DURATIONS

IV. U.S. EPA REGULATORY ACTIONS

IV.A. CLEAN AIR ACT (CAA)

No data available

IV.F. RESOURCE CONSERVATION AND RECOVERY ACT (RCRA)

IV.F.1. RCRA APPENDIX IX, for Ground Water Monitoring

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

(PROP) Physical-Chemical Properties:

V.B. PHYSICAL-CHEMICAL PROPERTIES

Chemical Formula -- Ni

Molecular Weight -- 58.70

Boiling Point -- 5139F, 2837C (Merck, 1983)

Specific Gravity (H2O=1) -- 8.90 (Sax, 1979)

Vapor Pressure (mmHg) -- 1 at 1810C (Sax, 1979)

Melting Point -- 2831F, 1555C (Merck, 1983)

Vapor Density (AIR=1) -- Not Found

Evaporation Rate (Butyl acetate=1) -- Not Found

Solubility in Water -- Insoluble (Weast, 1979)

Flash Point (Method Used) -- Not Found

Flammable Limits -- Not Found

Appearance and Odor -- Silvery metal (Weast, 1979); lustrous white metal (Merck, 1983)

Conditions or Materials to Avoid -- Finely divided nickel (e.g. Raney nickel catalysts) may become hot enough to ignite if exposed to air or moisture (Student, 1981, p. 363). Materials containing potassium perchlorate with nickel and titanium powders and infusional earth give severe explosions during a friction test. Dioxane reacts explosively with hydrogen and Raney nickel above 210C (NFPA, 1978). Also, aluminum; aluminum trichloride; ethylene; hydrogen; methanol; non-metals; oxidants; sulfur compounds (Sax, 1984, p. 1990), and selenium metal (Weiss, 1980, p. 1105) are incompatible with nickel.

Hazardous Decomposition or Byproducts -- Not Found

Use -- Nickel is used in nickel-plating; for various alloys such as new silver, Chinese silver, and German silver; for coins, electrotypes, lighting-rod tips, electrical contacts and electrodes, spark plugs, machinery parts; as a catalyst for hydrogenation of organic substances; in manufacturing of Monel metal, stainless steels, and nickel-chrome resistance wire; and in alloys for electronic and space applications (Merck, 1983).

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AFIS Accession Number 1076

(CAS) CAS Registry Number: 14797-55-8  
 (MAT) Material Name: Nitrate  
 (SYN) Synonyms: Nitrate;  
 Nitric acid, ion(1-)  
 (UPD) Update Date: 10-01-91  
 (EFF) Effective Date: 10-01-91  
 (STAT) Status:  
 STATUS OF DATA FOR Nitrate

File On-Line 01-31-87

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	10-01-91
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	pending	
Drinking Water Health Advisories (III.A.)	on-line	06-01-91
U.S. EPA Regulatory Actions (IV.)	on-line	03-01-88
Supplementary Data (V.)	no data	

(HAZ) Chronic Health Hazards, Noncarcinogenic:

(HAZO) Hazards Oral:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Early clinical signs of methemoglobinemia	NOAEL: 10 mg nitrate-nitrogen/L (1.6 mg/kg/day)	1	1	1.6E+0 mg/kg/day
in excess of 10% (0-3 months old infants formula)	LOAEL: 11-20 mg nitrate-nitrogen/L (1.8-3.2 mg/kg/day)			
Human Epidemiological Surveys				

Bosch et al., 1950;  
 Walton, 1951

\*Conversion Factor: Expressed as the amount of nitrogen within the nitrite molecule commonly shown as mg nitrate-nitrogen/L (1 mg nitrate-nitrogen = 4.4 mg nitrate). Doses based on ingestion of drinking water used to prepare infants' formula: 0.64 L/day by a 4 kg infant (0.16 L/kg/day) (Davidson et al., 1975). 10 mg/L x 0.64 L/day divided by 4 kg = 1.6 mg/kg/day.

#### I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Bosch, H.M., A.B. Rosefield, R. Huston, H.R. Shipman and F.L. Woodward. 1950. Methemoglobinemia and Minnesota well supplies. J. Am. Water Works Assoc. 42: 161-170.

Walton, G. 1951. Survey of literature relating to infant methemoglobinemia due to nitrate-contaminated water. Am. J. Public Health. 41: 986-996.

Most cases of infant methemoglobinemia are associated with exposure to nitrate in drinking water used to prepare infants' formula at levels >20 mg/L of nitrate-nitrogen (Bosch et al., 1950; Walton, 1951; Sattelmacher, 1962; Simon et al., 1964; ECETOC, 1988). Cases reported at levels of 11-20 mg/L nitrate-nitrogen are usually associated with concomitant exposure to bacteriologically contaminated water or excess intake of nitrate from other sources.

Bosch et al. (1950) evaluated 139 cases of cyanosis due to methemoglobinemia reported by physicians in Minnesota. All of the cases were in young children (ages 8 days to 5 months), with 90% occurring in infants <2 months of age. A study of the nitrate concentration of the wells (a total of 129) used to supply water to the children with methemoglobinemia was performed. None of the wells contained <10 mg/L nitrate-nitrogen. Two wells (1.5%) contained 10-20 mg/L, although the diagnosis of methemoglobinemia was considered questionable in both these cases. There were 25 wells (19%) that contained 21-50 mg/L, 53 (41%) that contained 51-100 mg/L, and 49 (38%) that contained >100 mg/L nitrate-nitrogen. Nearly all the wells were shallow with inadequate protection from surface contamination. Coliform organisms were detected in 45 of 51 samples (88%) tested for bacterial contamination.

Walton (1951) described a survey performed by the American Public Health Association to identify clinical cases of infantile methemoglobinemia that were associated with ingestion of nitrate-contaminated water. A total of 278

cases of methemoglobinemia were reported. Of 214 cases for which data were available on nitrate levels in water, none occurred in infants consuming water containing <10 mg nitrate-nitrogen/L (1.6 mg nitrate-nitrogen/kg/day). There were 5 cases (2%) in infants exposed to 11-20 mg nitrate-nitrogen/L (1.8-3.2 mg/kg/day), 36 cases (17%) in infants exposed to 21-50 mg/L (3.4-8.0 mg/kg/day), and 173 (81%) in infants exposed to >50 mg/L (>8 mg/kg/day). Data on the ages of the infants were not provided.

Cornblath and Hartmann (1948) supplied nitrate-containing water to eight healthy infants (ages 2 days to 11 months) at doses of 50 or 100 mg NO<sub>3</sub>/kg/day (11 or 23 mg nitrate-nitrogen/kg/day). Assuming average consumption of about 0.16 L/kg/day, this corresponds to concentrations of 70 or 140 mg nitrate-nitrogen/L. No cyanosis was evident in any infant, and the highest concentration of methemoglobin was 7.5%. These authors also administered doses of 100 mg/kg of nitrate to four healthy infants (age 2 days to 6 months) and to two infants (age 6 and 7 weeks) who had been admitted to the hospital for cyanosis. No cyanosis was produced in the healthy infants, but cyanosis did occur in the individuals with a prior history of cyanosis. Examination of the saliva, gastric juice and stools of these infants revealed the presence of bacteria that readily reduced nitrate to nitrite. The gastric pH of these infants was >4 in both cases.

Donahoe (1949) reported five cases of moderate to severe cyanosis in infants (age 1-7 weeks) in South Dakota. In four of the five cases, the water used to feed the infants was from shallow wells and was shown to be heavily contaminated with bacteria. Nitrate levels were measured in two cases, with values of 50 and 177 mg/L (12 and 41 mg nitrate-nitrogen/L), respectively. This corresponds to doses of 8 and 28 mg nitrate-nitrogen/kg/day.

Simon et al. (1964) measured methemoglobin levels in 89 healthy infants who received nitrate-free water, 38 infants who received water containing 11-23 mg nitrate-nitrogen/L (1.8-3.7 mg nitrate-nitrogen/kg/day), and 25 infants receiving water containing >23 mg nitrate-nitrogen/L (>3.7 mg nitrate-nitrogen/kg/day). For infants age 1-3 months, mean methemoglobin levels in these three groups were 1.0, 1.3 and 2.9%, respectively. For infants age 3-6 months, values were 0.8, 0.8 and 0.7%, respectively. No clinical signs of

methemoglobinemia were detected in any of the infants.

Toussaint and Selenka (1970) supplied 34 healthy infants (age 1-3 months) with formula prepared with water containing 150 mg nitrate/L (34.5 mg nitrate-nitrogen/L, corresponding to 5.5 mg nitrate-nitrogen/kg/day). Average methemoglobin levels rose from about 1% to about 2-3% within 1-2 days, and then tended to stay steady for up to 10 days. No clinical signs of methemoglobinemia were reported.

#### I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF - 1. An uncertainty factor of 1 was employed because available data define the no-observed-adverse-effect level for the critical toxic effect in the most sensitive human subpopulation.

MF - 1.

#### I.A.4. ADDITIONAL STUDIES / COMMENTS (ORAL RfD)

Nitrate toxicity is due primarily to its conversion to nitrite, which oxidizes the Fe(+2) form of iron in hemoglobin to the Fe(+3) state. This compound (methemoglobin) does not bind oxygen, resulting in reduced oxygen transport from lungs to tissues. Low levels of methemoglobin occur in normal individuals, with typical values usually ranging from 0.5 to 2.0% (NAS, 1981). However, due to the large excess capacity of blood to carry oxygen, levels of methemoglobin up to around 10% are not associated with any significant clinical signs (Walton, 1951; ECETOC, 1988). Concentrations above 10% may cause a bluish color to skin and lips (cyanosis), while values above 25% lead to weakness, rapid pulse and tachypnea (Jones et al., 1973). Death may occur if methemoglobin values exceed 50-60%.

Conversion of nitrate to nitrite is mostly mediated by bacteria in the gastrointestinal system. Consequently, the risk of methemoglobinemia from ingestion of nitrate depends not only on the dose of nitrate, but also on the number and type of enteric bacteria. In healthy adults, available data suggest about 5% of a dose of nitrate is reduced to nitrite by bacteria in the mouth (NAS, 1981). Conversion of nitrate to nitrite may also occur in the stomach if the pH of the gastric fluid is sufficiently high (above pH 5) to permit bacterial growth. This is of concern in adults with diseases such as achlorhydria or atrophic gastritis. It is also of concern in infants, since the infant gastrointestinal system normally has a high pH that favors the

growth of nitrate-reducing bacteria. For this reason, infants (especially age 0-3 months) are generally recognized as being the subpopulation most susceptible to nitrate-induced methemoglobinemia. Risk is especially high in infants who are exposed to water that is contaminated with bacteria, since this tends to promote high concentrations of bacteria in the stomach and intestines.

Nitrate is a normal component of the human diet. A typical daily intake by an adult in the United States is about 75 mg/day (about 0.2-0.3 mg nitrate-nitrogen/kg/day) (NAS, 1981). Of this, over 85% comes from the natural nitrate content of vegetables such as beets, celery, lettuce and spinach. Daily intakes of nitrate by vegetarians may exceed 250 mg/day (0.8 mg nitrate-nitrogen/kg/day) (NAS, 1981). The contribution from drinking water is usually quite small (about 2-3% of the total) (NAS 1981), but could reach 85 mg/day (0.29 mg nitrate-nitrogen/kg/day) if water containing 10 mg nitrate-nitrogen/L was consumed. Thus, some adults consuming high levels of vegetables along with water containing high levels of nitrate (up to 10 mg nitrate-nitrogen/L) could receive total doses of nitrate approaching the RfD of 1.6 mg nitrate-nitrogen/kg/day.

Two epidemiological studies have been performed on the adverse effects of nitrate exposure, but the results are internally inconsistent or inconclusive. Dorsch et al. (1984) found a statistically significant increase in risk of birth defects in children of women consuming groundwater (which contained 5-15 mg/L of nitrate) compared with women consuming rainwater (which contained <5 mg/L nitrate). These authors emphasized that their results are limited by a number of factors, and stated that "it would be premature to interpret our case-control findings exclusively in terms of water nitrate exposure." Arbuckle et al. (1988) reported nonstatistically significant increase in the odds ratio for birth defects in children of women exposed to well-water (26 mg/L nitrate, equivalent to 0.2 mg nitrate-nitrogen/kg/day) compared with rain water (0.1 mg/L nitrate, equivalent to 0.0008 mg nitrate-nitrogen/kg/day). However, decreased odds ratios (also not statistically significant) were noted for exposure to nitrate in spring water (17 mg/L, equivalent to 0.13 mg nitrate-nitrogen/kg/day) or public water (26 mg/L).

Craun et al. (1981) conducted an epidemiologic study of 102 children aged 1-8 years in Washington County, Illinois. Sixty-four children were selected



from families consuming high-nitrate water (22-111 mg/L nitrate-nitrogen) and 38 children (controls) were from families consuming water containing <10 mg/L nitrate-nitrogen. Ingestion of high-nitrate water was not found to result in above-normal methemoglobin levels in exposed children. Assuming ingestion of 0.1 L/kg/day by older children, these concentrations correspond to doses of 2.2-11 mg nitrate-nitrogen/kg/day. This study indicates that older children are much less susceptible to nitrate-induced methemoglobinemia than are infants.

The Food and Drug Administration sponsored extensive tests of the reproductive and developmental effects of  $\text{NaNO}_3$  and  $\text{KNO}_3$  in mice, rats, hamsters and rabbits (FDA, 1972a,b). Groups of 20-26 mice, rats or hamsters and 10-13 rabbits were treated by gavage on days 6-15 (mice, rats), days 6-10 (hamster) or days 6-18 (rabbits) of gestation. Fetuses were delivered by Cesarean section and examined for visceral and skeletal malformations. Dose levels (expressed as mg nitrate-nitrogen) ranged from 0.6-66 mg/kg/day for mice, from 0.3-41 mg/kg/day for rats, from 0.4-66 mg/kg/day for hamsters and from 0.3-41 mg/kg/day for rabbits. No significant effects were detected regarding maternal reproductive parameters (percent pregnant, abortion frequency, number of litters), fetotoxicity (percent fetal resorptions, live fetuses per dam, average fetal weight) or fetal malformations up to the maximum doses administered to each species. These studies identify a reproductive/developmental NOAEL of 66 mg nitrate-nitrogen/kg/day for mice and hamsters and 41 mg nitrate-nitrogen/kg/day for rats and rabbits.

Sleight and Atallah (1968) studied the effects of nitrate on reproduction and development in guinea pigs. Groups of 3-6 females were exposed to drinking water containing 0, 300, 2500, 10,000 or 30,000 ppm  $\text{KNO}_3$  for 143-204 days. This resulted in average doses of 0, 12, 102, 507 or 1130 mg nitrate-nitrogen/kg/day. Normal conception occurred at all dose levels. No significant effect on reproductive performance was detected except in the high-dose group, where there was a decrease in number of live births. The authors attributed the fetotoxic effects to hypoxia due to maternal methemoglobinemia, although data on this were not provided. No fetal malformations were observed at any dose. This study identifies a reproductive NOAEL of 507 and a LOAEL of 1130 mg nitrate-nitrogen/kg/day.

No multi-generation studies were located on the reproductive effects of nitrate. In the absence of such data, observations from animals exposed to

nitrite may be used as a conservative estimate of nitrate toxicity.

Hugot et al. (1980) performed a three-generation study in rats. Female animals were administered sodium nitrite in the diet at doses of 90 or 160 mg nitrite-nitrogen/kg/day. There were no effects on a number of reproductive parameters. Some pups showed small decreases in birth weight and growth rate during lactation, and changes in organ weights at weaning. This study identifies a LOAEL of 90 mg nitrite-nitrogen/kg/day. Assuming that a maximum of 10% of a dose of nitrate is converted to nitrite by an adult human, this would correspond to a LOAEL of 900 mg nitrate-nitrogen/kg/day.

Druckrey et al. (1963) supplied rats with  $\text{NaNO}_2$  in drinking water for three generations at a dose level of 100 mg/kg/day (20 mg nitrite-nitrogen/kg/day). No teratogenic effects or adverse effects on reproduction were detected in any generation. Assuming that a maximum of 10% of a dose of nitrate is converted to nitrite by an adult human, this would correspond to a NOAEL of 200 mg nitrate-nitrogen/kg/day.

No studies were located on systemic effects of nitrate in humans or animals. In the absence of such data, observations from animals exposed to nitrite may be used as a conservative estimate of nitrate toxicity. Druckrey et al. (1963) exposed rats for their lifetime to  $\text{NaNO}_2$  in drinking water at a dose of 100 mg/kg/day (20 mg nitrite-nitrogen/kg/day). No treatment-related histologic or hematologic effects were noted except for elevated methemoglobin levels in the treated animals.

Til et al. (1988) supplied rats with drinking water containing up to 3000 mg/L of  $\text{KNO}_2$  (500 mg nitrite-nitrogen/L, equivalent to 50 mg nitrite-nitrogen/kg/day) for 13 weeks. No histological effects were detected except for a very slight to slight hypertrophy of the zona glomerulosa. This was probably due to reduced water intake, and is not judged to constitute an adverse health effect. This study identifies a NOAEL of 17 and a LOAEL of 50 mg nitrite-nitrogen/kg/day (based on methemoglobin levels). Assuming that a maximum of 10% of a dose of nitrate is converted to nitrite by an adult human, this would correspond to a NOAEL of 170 and a LOAEL of 500 mg nitrate-nitrogen/kg/day.

Shuval and Gruener (1972) exposed rats for 24 months to water containing

0, 100, 1000, 2000 or 3000 ppm of sodium nitrite (0, 2, 20, 40 or 60 mg nitrite-nitrogen/kg/day). Histological examination of the lungs revealed dilated bronchi, fibrosis and emphysema at 1000 ppm or above. Histological examination of the heart revealed an increased percentage of coronary arteries that were characterized as "thin and dilated." This effect appears to be at least partly due to the absence of coronary artery thickening and narrowing that normally occurs in aged rats, so it is not certain that these changes are inherently adverse. Based on effects on the lung, this study identifies a NOAEL of 2 and a LOAEL of 20 mg nitrite-nitrogen/kg/day. Assuming that a maximum of 10% of a dose of nitrate is converted to nitrite by an adult human, this would correspond to a NOAEL of 20 and a LOAEL of 200 mg nitrate-nitrogen/kg/day.

#### I.A.5. CONFIDENCE IN THE ORAL RfD

Study: High  
Data Base: High  
RfD: High

The studies of Bosch et al. (1950) and Walton (1951) provide convincing evidence that infantile methemoglobinemia does not occur at drinking water levels of 10 mg nitrate-nitrogen/L or less. This is supported by a large number of additional epidemiological and case studies in humans (e.g., Cornblath and Hartmann, 1948; Simon et al., 1964; Toussaint and Selenka, 1970; Craun et al., 1981; see U.S. EPA, 1990 for descriptions of additional studies).

#### I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation -- U.S. EPA, 1990

Agency Work Group Review: 11/21/85, 02/05/86, 02/26/86, 06/20/90, 07/25/90, 08/22/90

Verification Date: 08/22/90

#### I.A.7. EPA CONTACTS (ORAL RfD)

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Ken Bailey / OW -- (202)260-5535 / FTS 260-5535

(CAR) Carcinogenicity Assessment:

(CARW) Carcinogenicity Weight:

## II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

This substance/agent has been evaluated by the U.S. EPA for evidence of human carcinogenic potential. This does not imply that this agent is necessarily a carcinogen. The evaluation for this chemical is under review by an inter-office Agency work group. A risk assessment summary will be included on IRIS when the review has been completed.

(HA) Hazard Assessment:

(HAS) Health Advisories (Drinking Water):

## III. HEALTH HAZARD ASSESSMENTS FOR VARIED EXPOSURE DURATIONS

### III.A. DRINKING WATER HEALTH ADVISORIES

#### III.A.1. ONE-DAY HEALTH ADVISORY FOR A CHILD

Appropriate data for calculating a One-day HA are not available. It is recommended that the Ten-day HA values (calculated below) be used as the One-day HA values.

#### III.A.2. TEN-DAY HEALTH ADVISORY FOR A CHILD

In developing the nitrate/nitrite HA it was determined that the 4-kg infant is the most sensitive member of the population with respect to both nitrate and nitrite. This determination was based on studies by Walton (1951) and Craun, et al. (1981). Walton (1951) reported over 278 cases of cyanosis and, in some cases, mortality in infants associated with the consumption of water containing greater than 10 mg/L nitrate-nitrogen. In relation to populations other than the 4-kg infant, Craun et al. (1981) reported that ingestion of water containing 22 to 111 mg/L nitrate-nitrogen by children aged 1 to 8 years did not result in abnormal mean methemoglobin levels and was not related to increased methemoglobin levels in comparison to controls.

Therefore, while the Ten-day HA is usually derived for the 10-kg child, in this case this value was derived for the most sensitive members of the population (4-kg infants), as well as for other populations.

Ten-day HA for 4-kg infant -- 1E+1 mg/L nitrate-nitrogen

NOAEL -- 10 mg/L

UF -- 1 (used for human study in sensitive subpopulations)

Assumptions -- none

Principal study -- Walton, 1951

More than 278 cases of cyanosis, and in some cases, mortality were associated with consumption of nitrate-contaminated water by the infant. No cases associated with water containing 10 mg/L or less of nitrate-nitrogen were found.

Ten-day HA for a 10-kg child --  $1 \times 10^2$  mg/L nitrate-nitrogen

NOAEL -- 111 mg/L

UF -- 1 (used for human study in sensitive subpopulations)

Assumptions -- none

Principal Study -- Craun et al., 1981

In an epidemiologic study of 102 children aged 1 to 8 years, 64 of the study subjects consumed water with high nitrate levels (22 to 111 mg/L nitrate-nitrogen) and 38 consumed water with low nitrate levels (<10 mg/L nitrate-nitrogen). Ingestion of water containing 22 to 111 mg/L nitrate-nitrogen did not result in abnormal mean methemoglobin levels and was not related to increased methemoglobin levels in comparison to controls. In the

entire study group of 102 children, only five had methemoglobin levels >2% (maximum of 3.1% in a child from the low exposure group).

#### III.A.3. LONGER-TERM HEALTH ADVISORY FOR A CHILD

Appropriate data for calculating Longer-term HAs are not available. However, as previously discussed, the 4-kg infant is the most sensitive member of the population with respect to the formation of methemoglobin induced by either nitrite directly or by the in vivo reduction of nitrate to nitrite. In addition, as the 4-kg infant ages (e.g., to a 10-kg child), sensitivity to the effects of methemoglobin as well as the amount of nitrate reduced to nitrite decreases, thus rendering the older child and adult less sensitive to the effects of both nitrate and nitrite. Thus, it has been concluded that the Ten-day HA for the 4-kg infant for nitrate-nitrogen (10 mg/L) will offer adequate protection against methemoglobin formation in all other age groups as well.

#### III.A.4. LONGER-TERM HEALTH ADVISORY FOR AN ADULT

Appropriate data for calculating Longer-term HAs are not available. See explanation in III.A.3.

#### III.A.5. DRINKING WATER EQUIVALENT LEVEL / LIFETIME HEALTH ADVISORY

Appropriate data for calculating a DWEL or a Lifetime HA are not available.

### III.A.7. ANALYTICAL METHODS FOR DETECTION IN DRINKING WATER

Determination of nitrite alone, or nitrite and nitrate combined, is by colorimetry or spectrophotometry.

### III.A.8. WATER TREATMENT

Treatment techniques which are capable of removing nitrates from drinking water include ion exchange and reverse osmosis.

### III.A.9. DOCUMENTATION AND REVIEW OF HAS

U.S. EPA. 1985. Final Draft of the Drinking Water Criteria Document on Nitrates/Nitrites. Office of Drinking Water, Washington, DC.

EPA review of HAS in 1985.

Public review of HAS following notification of availability in October, 1985.

Scientific Advisory Panel review of HAS in January, 1986.

Preparation date of this IRIS summary -- 06/17/87

### III.A.10. EPA CONTACTS

Kenneth Bailey / OW -- (202)260-5535 / FTS 260-5535

Edward V. Ohanian / OW -- (202)260-7571 / FTS 260-7571

(REGS) Regulations:

(SDWA) Safe Drinking Water Act:

#### IV. U.S. EPA REGULATORY ACTIONS

##### IV.B. SAFE DRINKING WATER ACT (SDWA)

##### IV.B.1. MAXIMUM CONTAMINANT LEVEL GOAL (MCLG) for Drinking Water

Value (status) -- 10.0 mg/L (Proposed, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- The MCLGs of 10.0 mg/L for nitrate/nitrogen and 1 mg/L for nitrite/nitrogen are proposed based on provisional DWELs of 10 mg/L (nitrate/nitrogen) and 1 mg/L (nitrite/nitrogen). A DWEL for nitrate/nitrogen was calculated from a NOAEL of 10 mg/L for methemoglobinemia in infants (epidemiologic study) with an uncertainty factor of 1. A DWEL for nitrite/nitrogen was calculated from the same NOAEL (10 mg/L) but with an uncertainty factor of 10 (because of direct toxicity).

Reference -- 50 FR 46936 Part IV (11/13/85)

EPA Contact -- Kenneth Bailey / Criteria and Standards Division, ODW /

(202)382-7571 / FTS 382-7571; or Drinking Water Hotline / (800)426-4791

IV.B.2. MAXIMUM CONTAMINANT LEVEL (MCL) for Drinking Water

Value (status) -- 10 mg/L (as nitrogen)

Considers technological or economic feasibility? -- NO

Discussion --

Reference -- 45 FR 57332 Part IV (08/27/80)

EPA Contact -- Kenneth Bailey / Criteria and Standards Division, ODW /  
(202)382-7571 / FTS 382-7571; or Drinking Water Hotline / (800)426-4791

Captured 4/17/92

1 - IRIS  
IRSN - 75  
DATE - 920120  
UPDT - 01/20/92, 52 fields  
STAT - Oral RfD Assessment (RDO) on-line 08/01/91  
STAT - Inhalation RfC Assessment (RDI) no data  
STAT - Carcinogenicity Assessment (CAR) pending  
STAT - Drinking Water Health Advisories (DWHA) on-line 03/01/88  
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92  
IRH - 03/31/87 RDO Documentation corrected  
IRH - 09/30/87 EXSR Regulatory Action section on-line  
IRH - 03/01/88 RDO Text added  
IRH - 03/01/88 HADV Health Advisory added  
IRH - 08/01/91 RDO Oral RfD summary noted as pending change  
IRH - 08/01/91 REFS Bibliography on-line  
IRH - 01/01/92 EXSR Regulatory actions updated  
RLEN - ND  
NAME - Nitrite  
RN - 14797-65-0  
SY - Nitrite  
SY - Nitrous acid, ion(1-)

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RDO -

o ORAL RFD SUMMARY :

NOTE: The oral RfD for nitrite may change in the near future pending the outcome of a further review now being conducted by the RfD/RfC Work Group.

Critical Effect	Experimental Doses*	UF	MF	RfD
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Methemoglobinemia	NOEL: 10 ppm of drinking water or 10 mg/L converted to 1.0 mg/kg/day	1	10	1E-1 mg/kg/day
Infant Chronic Exposure to Drinking Water				

Walton, 1951                      LOAEL: 11-20 ppm

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\*Conversion Factor: 1 L drinking water/day 10 kg child; thus, 10 mg/L x 1 L/day / 10 kg = 1.0 mg/kg/day

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o ORAL RFD STUDIES :

Walton, G. 1951. Survey of literature relating to infant methemoglobinemia due to nitrate-contaminated water. Am. J. Public Health. 41: 986-996.

This is an epidemiologic study on the incidence of methemoglobinemia in infants routinely fed formula prepared from nitrate-contaminated water. This study analyzed all known cases of infant methemoglobinemia occurring in 37 U.S. states irrespective of date or type of water supply. Nitrate (nitrogen) content ranged from 10 ppm to over 100 ppm. No incidences of methemoglobi-



nemia were found to occur in drinking water containing greater than 10 ppm (10 mg/L) nitrate (nitrogen). A NOEL of 10 mg/L was derived from these studies.

Exposure of hemoglobin to nitrite results in the oxidation of the hemoglobin to methemoglobin. Animals do not provide a good model for methemoglobin formation because many species lack nitrate-reducing bacteria. Infants are, however, particularly susceptible due to their high gut content of nitrate-reducing bacteria, their lower enzymatic capacity to reduce methemoglobin to hemoglobin, and to the the presence of hemoglobin F, which is more susceptible to oxidation.

Several more recent studies support Walton's (1951) 10 mg/L NOEL for infant methemoglobinemia (NAS, 1977; Winton, 1971; Calabrese, 1978).

Using the NOEL from the Walton study and a modifying factor of 10, the RfD for nitrite was calculated (U.S. EPA, 1985) for a 10-kg child drinking 1 L of water/day as 0.1 mg/kg/day or 1 mg/day.

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o ORAL RFD UNCERTAINTY :

UF = 1. No uncertainty factor was used in the derivation of the RfD because the NOEL was of the critical toxic effect (i.e., methemoglobinemia) in the sensitive human population (i.e., infants). The length of exposure encompassed both the critical effect and the sensitive population.

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o ORAL RFD MODIFYING FACTOR :

MF = 10. A modifying factor of 10 was applied because of the direct toxicity of nitrite.

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o ORAL RFD COMMENTS :

An RfD of 0.2 mg/kg/day could be calculated from the Walton (1951) study using the body weight of 4 kg and fluid consumption of 0.64 L/day for infants. The lower value of 0.1 mg/kg/day is maintained, however, because of the uncertainties in the changing fluid consumption and body weight as a neonate (4 kg) ages to a 2-year-old child (10 kg). While there are some data to the contrary, it is most likely that older children do not respond with increased methemoglobin to nitrate in drinking water. For example, Craun et al. (1981) reported that 64 children aged 1-8, consuming water with nitrate nitrogen concentrations of 22 to 111 mg/L, had an average methemoglobin concentration of 1.13%. This is not considered to be elevated and was in fact no different from the level (0.98%) observed in 38 children who drank water contaminated with less than 10 mg nitrate/L.

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o ORAL RFD CONFIDENCE :

Study: High  
Data Base: High  
RfD: High

Confidence in the study is high because the NOEL is determined in the known sensitive human population. The data base contains several recent supporting epidemiologic studies for the critical effect in the sensitive population (infants); therefore, a high confidence rating is given to the data base. High confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

The only U.S. EPA documentation at present is on IRIS.

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o REVIEW DATES : 11/21/85, 02/05/86, 02/26/86  
o VERIFICATION DATE : 02/26/86  
o EPA CONTACTS :

Kenneth L. Bailey / ODW -- (202)260-5535 / FTS 260-5535

Rita S. Schoeny / ORD -- (513)569-7814 / FTS 684-7814

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HAONE-

Appropriate data for calculating a One-day HA are not available. It is recommended that the Ten-day HA values (calculated below) be used as the One-day HA values.

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HATEN-

NOTE: In developing the nitrate/nitrite HA it was determined that the 4-kg infant is the most sensitive member of the population with respect to both nitrate and nitrite. This determination was based on studies by Walton (1951) and Craun, et al. (1981). Walton (1951) reported over 278 cases of cyanosis and, in some cases, mortality in infants associated with the consumption of water containing greater than 10 mg/L nitrate-nitrogen. In relation to populations other than the 4-kg infant, Craun et al. (1981) reported that ingestion of water containing 22 to 111 mg/L nitrate-nitrogen by children aged 1 to 8 years did not result in abnormal mean methemoglobin levels and was not related to increased methemoglobin levels in comparison with controls. Therefore, while the Ten-day HA is usually derived for the 10-kg child, in this case this value was derived for the most sensitive members of the population (4-kg infants), as well as for other populations.

Ten-day HA for 4-kg infant -- 1E+0 mg/L nitrite-nitrogen

NOAEL -- 10 mg/L

UF -- 1 (used for human study in sensitive subpopulations)  
Assumptions -- 10% conversion of nitrate to nitrite by 4-kg infant

Principal study -- Walton, 1951

More than 278 cases of cyanosis, and in some cases, mortality were associated with consumption of nitrate-contaminated water by the infant. No cases associated with water containing 10 mg/L or less of nitrate-nitrogen were found.

Ten-day HA for a 10-kg child -- 1E+1 mg/L nitrite-nitrogen

NOAEL -- 111 mg/L

UF -- 1 (used for human study in sensitive subpopulation)  
Assumptions -- 10% conversion of nitrate to nitrite by 10-kg child

Principal Study -- Craun et al., 1981

In an epidemiologic study of 102 children aged 1 to 8 years, 64 of the study subjects consumed water with high nitrate levels (22 to 111 mg/L nitrate-nitrogen) and 38 consumed water with low nitrate levels (<10 mg/L nitrate-nitrogen). Ingestion of water containing 22 to 111 mg/L nitrate-nitrogen did not result in abnormal mean methemoglobin levels and was not related to increased methemoglobin levels in comparison to controls. In the entire study group of 102 children, only five had methemoglobin levels greater than 2% (maximum of 3.1% in a child from the low exposure group).

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HALTC-

Appropriate data for calculating Longer-term HAs are not available. However, as previously discussed, the 4-kg infant is the most sensitive member of the population with respect to the formation of methemoglobin induced by either nitrite directly or by the in vivo reduction of nitrate to nitrite. In addition, as the 4-kg infant ages (e.g., to a 10-kg child), sensitivity to the effects of methemoglobin as well as the amount of nitrate reduced to nitrite decreases, thus rendering the older child and adult less sensitive to the effects of both nitrate and nitrite. Thus, it has been concluded that the Ten-day HA for the 4-kg infant for nitrite-nitrogen (1 mg/L) will offer adequate protection against methemoglobin formation in all other age groups as well.

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HALTA-

Appropriate data for calculating Longer-term HAs are not available.  
See explanation in HALTC

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HALIF-

Appropriate data for calculating a DWEL or a Lifetime HA are not available. See explanation in HALTC

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OLEP -

No data available

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ALAB -

Determination of nitrite alone, or nitrite and nitrate combined, is by colorimetry or spectrophotometry.

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TREAT-

Treatment techniques which are capable of removing nitrates from drinking water include ion exchange and reverse osmosis.

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HADR -

o HEALTH ADVISORY SOURCE :

Walton, G. 1951. Survey of literature relating to infant methemoglobinemia due to nitrate-contaminated water. Am. J. Pub. Health. 41: 986-996.

DOCUMENT

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o HEALTH ADVISORY REVIEW :

Craun, G.F., D.G. Greathouse and D.H. Gunderson. 1981. Methemoglobin levels in young children consuming high nitrate well water in the United States. Int. J. Epidemiol. 10: 309-317.

U.S. EPA. 1985. Final Draft of the Drinking Water Criteria Document on Nitrates/Nitrites. Office of Drinking Water, Washington, DC.

EPA review of HAs in 1985.

Public review of HAs following notification of availability in October, 1985.

Scientific Advisory Panel review of HAs in January, 1986.

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o EPA DRINKING WATER CONTACT :

Kenneth Bailey / ODW -- (202)260-5535 / FTS 260-5535

Edward V. Ohanian / ODW -- (202)260-7571 / FTS 260-7571

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WQCHU-

No data available  
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WQCAQ-

Freshwater:

Acute -- none  
Chronic -- none

Marine:

Acute -- none  
Chronic -- none

Considers technological or economic feasibility? -- NO

Discussion -- Recognizing that concentrations of nitrate/nitrite that would exhibit toxic effects on fish could rarely occur in nature, restrictive criteria were not recommended.

Reference -- Quality Criteria for Water, EPA 440/9-76-023 (7/76), PB-263943.

EPA Contact -- Criteria and Standards Division / OWRS  
(202)260-1315 / FTS 260-1315

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MCLG -

Value (status) -- 1 mg/L [as nitrogen] (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion -- Nitrite has been classed a Category III contaminant with methemoglobinemia in infants identified as the most sensitive endpoint. The MCLG of 1.0 mg/L for nitrite/nitrogen is based on a review of all available data that demonstrates that 1 mg/L is adequate to protect infants and all other groups against the non/oncogenic effects of nitrite in drinking water. The MCLG is based upon a DWEL for nitrite/nitrogen of 1 mg/L.

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Health and Ecological Criteria Division / OST /  
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

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MCL -

Value -- 1.0 mg/L [as nitrogen] (Final, 1991)

Considers technological or economic feasibility? -- YES

Discussion -- The EPA has promulgated an MCL equal to the MCLG of 1.0 mg/L.

Monitoring requirements -- All systems must take one sample between 1993-1995.

Analytical methodology -- Spectrophotometric (EPA 354.1; SM 419); automated cadmium reduction (EPA 353.2; SM 418F; ASTM D-3867-79A); manual cadmium reduction (EPA 353.3; SM 418C; ASTM D-3867-79B0; ion chromatography (EPA 300; SM 429; ASTM D-4327-88); PQL= 0.4 mg/L.

Best available technology -- Ion exchange; reverse osmosis.

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW /  
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

#### \_\_\_IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

#### \_\_\_IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

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TSCA -

#### \_\_\_IV.E.1. TSCA, SECTION 6

Status -- Advance Notice of Proposed Rulemaking (ANPR) (1984)

Discussion -- EPA is proposing to investigate potential occupational risk for machinists from the formulation of nitrosamines when water-based metalworking fluids are combined with nitrite.

Reference: 49 FR 2767 (01/23/84); 40 CFR 747

EPA Contact -- Chemical Control Division / OTS (202)260-3749 / FTS 260-3749

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- OREF - Calabrese, E.J. 1978. Drinking Water Standards. In: Methodological Approaches to Deriving Environmental and Occupational Health Standards. John Wiley and Sons, Inc., New York, NY. p. 165-169.
- OREF - Craun, G.F., D.G. Greathouse and D.H. Gunderson. 1981. Methemoglobin levels in young children consuming high nitrate well water in the United States. Int. J. Epidemiol. 10: 309-317.
- OREF - NAS (National Academy of Sciences). 1977. Drinking Water and Health. Washington, DC.
- OREF - U.S. EPA. 1985. Drinking Water Criteria Document for Nitrates/Nitrites. Office of Drinking Water, Washington, DC.
- OREF - Walton, G. 1951. Survey of literature relating to infant methemoglobinemia due to nitrate-contaminated water. Am. J. Public Health. 41: 986-996.
- OREF - Winton, E.F., R.G. Tardiff and L.J. McCabe. 1971. Nitrate in drinking water. J. Am. Water Works Assoc. 63: 95-98.
- IREF - None
- CREF - None
- HAREF- Craun, G.F., D.G. Greathouse and D.H. Gunderson. 1981. Methemoglobin levels in young children consuming high nitrate well water in the United States. Int. J. Epidemiol. 10: 309-317.
- HAREF- Walton, G. 1951. Survey of literature relating to infant methemoglobinemia due to nitrate-contaminated water. Am. J. Pub. Health. 41: 986-996.
- HAREF- U.S. EPA. 1985. Drinking Water Criteria Document for Nitrates/Nitrites. Office of Drinking Water, Washington, DC.

Captured 8/12/92

1 - IRIS  
IRSN - 43  
DATE - 920120  
UPDT - 01/20/92, 52 fields  
STAT - Oral RfD Assessment (RDO) no data  
STAT - Inhalation RfC Assessment (RDI) no data  
STAT - Carcinogenicity Assessment (CAR) on-line 01/01/91  
STAT - Drinking Water Health Advisories (DWHA) no data  
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92  
IRH - 03/31/87 EXSR RQ added  
IRH - 09/30/87 EXSR Regulatory Action section on-line  
IRH - 03/01/88 CAREV Text clarified  
IRH - 03/01/88 CARO Number rounded off  
IRH - 03/01/88 CARO Text revised  
IRH - 03/01/88 CARO Confidence statement revised  
IRH - 03/01/88 CARI Number rounded off  
IRH - 03/01/88 CARI Confidence statement revised  
IRH - 03/01/88 CARDR Secondary contact changed  
IRH - 02/01/90 REFS Bibliography on-line  
IRH - 03/01/90 CREF Druckrey & Peto references clarified  
IRH - 01/01/91 CAR Text edited  
IRH - 01/01/91 CARI Inhalation slope factor removed (global change)  
IRH - 01/01/92 EXSR Regulatory actions updated  
RLEN - 12629  
NAME - N-Nitrosodimethylamine  
RN - 62-75-9  
SY - dimethylamine, N-nitroso  
SY - dimethylnitrosamin  
SY - Dimethylnitrosamine  
SY - dimethylnitrosoamine  
SY - DMNA: DMN  
SY - methylamine, N-nitrosodi-  
SY - NDMA  
SY - nitrosodimethylamine  
SY - Nitrosodimethylamine, N-  
SY - N-methyl-N-nitrosomethanamine  
SY - N,N-dimethylnitrosamine  
SY - N-Nitrosodimethylamine  
SY - RCRA waste number P082  
MF - C2H6N2O  
USE - Dimethylnitrosamine was formerly used in the production of rocket fuels. Dimethylnitrosamine is presently used as an antioxidant, as an additive for lubricants, and as a softener of copolymers (Merck, 1983, p. 952). It is an intermediate for 1,1-dimethylhydrazine (SRI, 1983).  
COFO - Yellow oily liquid; faint characteristic odor.  
ODOR - Yellow oily liquid; faint characteristic odor.  
BP - 304-307F, 151-153C  
MP - Not Found  
MW - 74.08  
DEN - 1.0048 at 20C/4C  
VAP - Not Found  
VAPD - Not Found  
EVAP - Not Found  
SOLW - Very soluble  
FLPT - Not Found  
FLMT - Not Found  
AVOI - Avoid exposure to ultraviolet light (Clayton and Clayton, 1981, p. 3119).  
DCMP - When heated to decomposition, it emits toxic fumes of nitrogen oxides (Sax, 1984, p. 1180-1181).  
CAREV-  
o CLASSIFICATION : B2; probable human carcinogen  
o BASIS FOR CLASSIFICATION : Induction of tumors at multiple sites in both rodents and nonrodent mammals exposed by various routes  
o HUMAN CARCINOGENICITY DATA :

Human exposure to nitrosamines results from contact with mixtures containing these compounds (e.g., cutting oils, tobacco products). Because of potential confounding by the other substances in these mixtures, data from



human exposure is of limited use in the evaluation of carcinogenicity of individual nitrosamines.

o ANIMAL CARCINOGENICITY DATA :

There is a large data base on the carcinogenicity of nitrosamines, most of which pertains to structure-activity relationships rather than to dose-response. N-Nitrosodimethylamine produced liver tumors in 80 rats when administered in drinking water (Druckrey et al., 1967) and in female Porton rats when administered in the diet (Terracini et al., 1967). Magee et al. (1976) state that dimethylnitrosamine produced many hemangiomatous tumors and some parenchymal cell tumors in the livers of rats after oral administration.

N-Nitrosodimethylamine acts as a transplacental carcinogen when administered to pregnant rats, mice, and Syrian golden hamsters by several routes (Tomatis, 1973). Increases in lung, liver, and kidney tumors were observed in both Wistar rats and Balb/C mice exposed by inhalation. Mink are very sensitive to the effects of dimethylnitrosamine, developing tumors when fed 0.05 mg/kg 2 days/week (NAS, 1978).

Peto et al. (1984) exposed groups of Colworth rats (36/sex/dose) to 15 concentrations of N-nitrosodimethylamine in drinking water (0.033-16.896 ppm). Daily water consumption was 41 mL/kg for males and 72 mL/kg for females. Tumors were generally of hepatic origin, and these tumors constituted the only cause of mortality considered treatment-related. Tumor incidences for each treatment group were not reported, but pooled data indicated possible positive trends for lung, skin, seminal vesicle, lymphatic/hematopoietic system, and liver tumors.

o SUPPORTING DATA :

N-Nitrosodimethylamine is mutagenic for *Escherichia coli*, *Salmonella typhimurium* and *Neurospora crassa*, produces mitotic recombination in *Sacharoyus cerevesiae*, recessive lethal mutations in *Drosophilla melanogaster*, and chromosomal aberrations in mammalian cells. Positive responses in bacterial cells are dependent upon the addition of a mammalian metabolism system (Montesano and Bartsch, 1976). Dimethylnitrosamine is structurally related to known carcinogens.

CARO -

- o CLASSIFICATION : B2; probable human carcinogen
- o BASIS FOR CLASSIFICATION : Induction of tumors at multiple sites in both rodents and nonrodent mammals exposed by various routes
- o ORAL SLOPE FACTOR :  $5.1E+1$  per (mg/kg)/day
- o DRINKING WATER UNIT RISK :  $1.4E-3$  per (ug/L)
- o DOSE EXTRAPOLATION METHOD : Weibull, extra risk
- o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	7E-2 ug/L
E-5 (1 in 100,000)	7E-3 ug/L
E-6 (1 in 1,000,000)	7E-4 ug/L

o ORAL DOSE-RESPONSE DATA :

Study reference: Species/strain; Tumor type; Route	Dose Administered Human Equivalent (mg/kg/day)	Tumor Incidence
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Peto et al., 1984: Rats/Colworth, female; liver tumors; drinking water	Specific tumor incidences were not published.	
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Data from Peto et al. (1984) on incidence of liver tumors of all types in female rats were shown to follow this relationship:

$$CI = 51.45 (d + 0.1)^{.6} \times t^{.7}$$

where: CI = cumulative incidence

d = dose (mg/kg/day)

t = time in years

Using procedures described in U.S. EPA (1980) to correct for background response, the increased risk of 1 ug/kg/day for 3 years =  $7.8E-3$  or a slope factor for rats of 7.8 per (mg/kg)/day. The slope factor was thus calculated to be 51 per (mg/kg)/day by using the cube root of the ratio of the assumed human body weight (70 kg) to the reported rat body weight of (250 g).

o ADDITIONAL COMMENTS :

The unit risk should not be used if the water concentration exceeds 7 ug/L, since above this concentration the unit risk may not be appropriate.

o DISCUSSION OF CONFIDENCE :

Although specific tumor incidence data was not reported, it appears that large numbers of animals were treated over a wide dose range. Both tumor incidence and latency were shown to be dose-dependent. The study was designed specifically for analysis using the Weibull model. A slope factor based on data by Druckrey et al. (1972) was determined by use of a one-hit model to be 26 per (mg/kg)/day.

CARI -

- o CLASSIFICATION : B2; probable human carcinogen
- o BASIS FOR CLASSIFICATION : Induction of tumors at multiple sites in both rodents and nonrodent mammals exposed by various routes
- o INHALATION UNIT RISK :  $1.4E-2$  per (ug/cu.m)
- o DOSE EXTRAPOLATION METHOD : Weibull, extra risk
- o RISK/AIR CONCENTRATIONS :

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	$7E-3$ ug/cu.m
E-5 (1 in 100,000)	$7E-4$ ug/cu.m
E-6 (1 in 1,000,000)	$7E-5$ ug/cu.m

o INHALATION DOSE-RESPONSE DATA :

Calculated from data in CARO.

o ADDITIONAL COMMENTS :

The above unit risk should not be used if the air concentration exceeds 0.7 ug/cu.m, since above this concentration the unit risk may not be appropriate.

o DISCUSSION OF CONFIDENCE :

See CARO.

CARDR-

o CARCINOGENICITY SOURCE :

U.S. EPA 1980. Ambient Water Quality Criteria for Nitrosamines. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-064. NTIS PB 81-117756.

U.S. EPA. 1986. Health and Environmental Effects Profile for Nitrosamines. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.

The values in the Health and Environmental Effects Profile for Nitrosamines (U.S. EPA, 1986) received Agency review.

DOCUMENT

o REVIEW DATES : 06/26/86, 08/13/86, 10/29/86  
o VERIFICATION DATE : 10/29/86  
o EPA CONTACTS :

James W. Holder / ORD -- (202)260-5721 / FTS 260-5721

Jim Cogliano / ORD -- (202)260-9243 / FTS 260-9243

ACUTE-

o ACUTE TOXICITY :

Dimethylnitrosamine is characterized as having extremely high toxicity (Sunshine, 1969). The lowest lethal oral dose in humans has been reported at 10 mg/kg/80 week intermittent exposure (NIOSH/RTECS, 1985).

o SIGNS AND SYMPTOMS :

Symptoms include nausea, vomiting, and malaise (Cooper, 1980). Chronic exposure may cause liver disease with jaundice and swelling (Hamilton, 1984) with low platelet count (Cooper, 1980).

WQCHU-

Water and Fish Consumption:  $1.4E-3$  ug/L

Fish Consumption Only:  $1.6E+1$  ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For maximum protection from potential carcinogenic effects because of exposure to dimethylnitrosamine, the ambient water concentrations should be zero. The criteria given represent an incremental risk of cancer over a lifetime of  $1.0E-6$ .

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OMS  
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute LEC --  $5.85E+3$  ug/L  
Chronic LEC -- None

Marine:

Acute LEC --  $3.3E+6$  ug/L  
Chronic LEC -- None

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LECs are given when the minimum data required to derive water quality criteria are not available. The values given represent nitrosamines as a class.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OURS  
(202)260-1315 / FTS 260-1315

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CERC -

Value (status) -- 10 pounds (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The RQ for dimethylnitrosamine is based on potential carcinogenicity. The available data indicate a hazard ranking of medium based on a potency factor of 25.55/mg/kg/day and the weight-of-evidence classification of B2. This corresponds to an RQ of 10 pounds.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)260-3000 / FTS 260-3000

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RCRA -

Status -- Listed

Reference -- 50 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)260-3000 / FTS 260-3000

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TSCA -

No data available

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OREF - None

IREF - None

CREF - Druckrey, H., R. Preussmann, S. Ivankovic and D. Schmechl. 1967. Organotropism and c carcinogenic effects of 65 different N-nitroso compounds in BD-rats. Z. Krebsforsch. 69(2): 103-201.

CREF - Druckrey, H., S. Ivankovic, R. Preussmann, K.J. Zulch and H.D. Mennel. 1972. Selective induction of malignant tumors of the nervous system by resorptive carcinogens. In: Experimental Biology of Brain Tumors. p. 85-112.

CREF - Magee, P.N., R. Montesano and R. Preussmann. 1976. N-nitroso compounds and related carcinogens. ACS Monograph. 173: 491-625.

CREF - Montesano, R. and H. Bartsch. 1976. Mutagenic and carcinogenic N-nitroso compounds: Possible environmental hazards. Mutat. Res. 32: 179-228.

CREF - NAS (National Academy of Sciences). 1978. Nitrates: An environmental assessment. A report prepared by the panel on nitrates of the Coordinating Comm. Sci. Tech. Assess. Environ. Pollut., Washington, DC.

CREF - Peto, R., R. Gray, P. Brantom and P. Grasso. 1984. Nitrosamine carcinogenesis in 5120 rodents: Chronic administration of sixteen different concentrations of NDEA, MDNA, MPYR and NPIP in the water of 4440 inbred rats, with parallel studies on NDEA alone of the effect of age of starting (3, 6 or 20 weeks) and of species (rats, mice, hamsters). IARC Sci. Publ. 57: 627-665.

CREF - Terracini, B., P.N. Magee and J.M. Barnes. 1967. Hepatic pathology in rats on low dietary levels of dimethylnitrosamine. Br. J. Cancer. 21: 559-565.

CREF - Tomatis, L. 1973. Transplacental carcinogenesis. In: Modern Trends in Oncology. Part I, R.W. Raven, Ed. Butterworths, London.

CREF - U.S. EPA 1980. Ambient Water Quality Criteria for Nitrosamines. Prepared by the Office of Health and Environmental Assessment,

Environmental Criteria and Assessment Office, Cincinnati, OH for the  
Office of Water Regulations and Standards, Washington, DC. EPA  
440/5-80-064. NTIS PB 81-117756.

CREF - U.S. EPA. 1986. Health and Environmental Effects Profile for  
Nitrosamines. Prepared by the Office of Health and Environmental  
Assessment, Environmental Criteria and Assessment Office, Cincinnati,  
OH for the Office of Solid Waste and Emergency Response, Washington,  
DC.

HAREF- None

IRIS Accession Number 1178

(CAS) CAS Registry Number: 86-30-6  
(MAT) Material Name: N-Nitrosodiphenylamine  
(SYN) Synonyms: BENZENAMINE, N-NITROSO-N-PHENYL-;  
CURETARD A;  
DELAC J;  
DIPHENYLAMINE, N-NITROSO-;  
DIPHENYLNITROSAMIN;  
DIPHENYLNITROSAMINE;  
DIPHENYL N-NITROSOAMINE;  
NAUGARD TJB;  
NCI-C02880;  
NDPA;  
NDPhA;  
NITROSODIPHENYLAMINE;  
Nitrosodiphenylamine, N-;  
NITROUS DIPHENYLAMIDE;  
N,N-DIPHENYLNITROSAMINE;  
N-NITROSODIFENYLAMIN;  
N-Nitrosodiphenylamine;  
N-NITROSO-N-PHENYLANILINE;  
REDAX;  
RETARDER J;  
TJB;  
VULCALENT A;  
VULCATARD;  
VULCATARD A;  
VULKALENT A;  
VULTROL

(UPD) Update Date: 03-01-88

(EFF) Effective Date: 10-01-91

(STAT) Status:

STATUS OF DATA FOR N-Nitrosodiphenylamine

File On-Line 03-31-87

Category (section)	Status	Last Revised
-----	-----	-----
Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	03-01-88
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	on-line	03-01-88
Supplementary Data (V.)	no data	

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(CAR) Carcinogenicity Assessment:

(CARW) Carcinogenicity Weight:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen

Basis -- Increased incidence of bladder tumors in male and female rats and reticulum cell sarcomas in mice, and structural relationship to carcinogenic nitrosamines

#### II.A.2. HUMAN CARCINOGENICITY DATA

Inadequate. Human exposure to nitrosamines results from contact with mixtures containing these compounds (e.g., cutting oils, tobacco products). Because of potential confounding by the other substances in these mixtures,

data are of limited use in the evaluation of carcinogenicity of individual nitrosamines.

#### II.A.3. ANIMAL CARCINOGENICITY DATA

N-nitrosodiphenylamine (98% pure containing two unspecified impurities) was administered at 0, 1000 or 4000 ppm in diet to groups of 50 F344 rats/sex. Matched controls consisted of 20 rats/sex. Dose-related mortality was

noted in females. Statistically increased incidence of urinary bladder transitional cell carcinomas was observed in both sexes. Epithelial hyperplasia and squamous metaplasia also occurred, as did integumentary fibromas in males (NCI, 1979).

In the same study no increased tumor incidence was observed in B6C3F1 mice receiving dietary doses of 10,000 and 20,000 ppm (males) or 2475 and 6139 ppm (TWA, females). Likewise, no evidence of carcinogenicity was observed in BD rats administered 120 mg nitrosodiphenylamine/kg in water for

541 days or in male Wistar rats gavaged with 1.07 mg/day in 1.1% aqueous methylcellulose 5 days/week for 45 weeks (Druckrey et al., 1967; Argus and Hoch-Ligeti, 1961). Neither B6C3F1 nor B6AKF1 mice showed statistically significant increases in tumor incidence following gavage with 1000 mg/kg/day from day 7-28 of age followed by dietary exposure to 3769 ppm until weeks 77-79 of life (BRL, 1968; Innes et al., 1969). Weekly topical application of diphenylnitrosoamine for 20 weeks did not induce tumors in hr/hr Oslo mice, nor did weekly i.p. injection of 2.5 mg in PEG 400 (Iverson, 1980; Boyland et al., 1968). A single s.c. injection of 1000 mg/kg/day resulted in significantly increased incidence of reticulum cell sarcomas in male B6C3F1 mice, but not in females or B6AKF1 mice of either gender (BRL, 1968).

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Nitrosodiphenylamine has produced mixed responses in genetic toxicology tests. It was negative in bacterial mutation assays, mutation assays in V79

and CHO and mouse lymphoma cells and SCE in CHO cells (IARC, 1982). Positive responses have been obtained for several endpoints in *S. cerevisiae* (de Serres and Hoffmann, 1981) and in DNA damage assays in rat hepatocytes (Althaus et al., 1982; Sina et al., 1983). N-nitrosodiphenylamine produced transformation of Syrian hamster embryo cells, BHK cells and F344 rat embryo

cells infected with Rauscher murine leukemia viruses (Pienta and Kawalek, 1981; Daniel and Dehnel, 1981; Dunkel et al., 1981).

N-nitrosodiphenylamine is structurally related to carcinogenic nitrosamines.

(CARO) Carcinogenicity Oral:

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor --  $4.9E-3/\text{mg/kg/day}$

Drinking Water Unit Risk --  $1.4E-7/\text{ug/L}$

Extrapolation Method -- Linearized multistage procedure, extra risk

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
-----	-----
E-4 (1 in 10,000)	$7E+2 \text{ ug/L}$
E-5 (1 in 100,000)	$7E+1 \text{ ug/L}$
E-6 (1 in 1,000,000)	$7E+0 \text{ ug/L}$

II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Species/Strain	Dose		Tumor	Reference
Tumor Type	Administered	Human Equivalent	Incidence	
-----				

Rat/F344, female; Route: Oral, diet

NCI, 1979

transitional  
cell carcinoma  
of the bladder

ppm	mg/kg/day	mg/kg/day	
0	0	0	0/18
1000	50	7.7	0/48
4000	200	30.6	40/49

II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)



The unit risk should not be used if the water concentration exceeds  $7E+4$  ug/L, since above this concentration the slope factor may differ from that stated.

#### II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

Adequate numbers of animals were treated and observed for their lifetime. Significant increases in tumor incidence were observed only in high-dose animals. NCI noted that the mechanism by which bladder tumors were induced (e.g., calculus formation or nitrosation of amines in feed) is not known.

(CARDOC) Carcinogenicity Documentation:

#### II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

##### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1986. Health and Environmental Effects Profile for Nitrosamines. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.

##### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The 1986 Health and Environmental Effects Profile for Nitrosoamines has received Agency review.

Agency Work Group Review: 02/11/87

Verification Date: 02/11/87

##### II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

James W. Coglianò / ORD -- (202)260-9243 / FTS 260-9243

Jim Holder / ORD -- (202)260-5721 / FTS 260-5721

(REGS) Regulations:

(CAA) Clean Air Act:

#### III. HEALTH HAZARD ASSESSMENTS FOR VARIED EXPOSURE DURATIONS

#### IV. U.S. EPA REGULATORY ACTIONS

##### IV.A. CLEAN AIR ACT (CAA)

No data available

(RCRA) Resource Conservation and Recovery Act:  
IV.F. RESOURCE CONSERVATION AND RECOVERY ACT (RCRA)  
IV.F.1. RCRA APPENDIX IX, for Ground Water Monitoring

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

Captured 4/17/92

1 - IRIS  
IRSN - 174  
DATE - 920122  
STAT - Oral RfD Assessment (RDO) no data  
STAT - Inhalation RfC Assessment (RDI) no data  
STAT - Carcinogenicity Assessment (CAR) on-line 03/01/88  
STAT - Drinking Water Health Advisories (DWHA) no data  
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92  
IRH - 03/01/88 CARO Confidence statement revised  
IRH - 03/01/88 CARDR Contacts switched  
IRH - 06/01/90 RCRA EPA contact changed  
IRH - 06/01/90 REFS Bibliography on-line  
IRH - 01/01/92 EXSR Regulatory actions updated

RLEN - ND

NAME - N-Nitrosodi-N-propylamine

RN - 621-64-7

SY - DIPROPYLAMINE, N-NITROSO-

SY - DIPROPYLNITROSAMINE

SY - DI-n-PROPYLNITROSAMINE

SY - DPN

SY - DPNA

SY - NDPA

SY - N-Nitrosodi-N-propylamine

SY - N-NITROSODIPROPYLAMINE

SY - N-NITROSODI-n-PROPYLAMINE

SY - N-NITROSO-N-PROPYL-1-PROPANAMINE

SY - PROPANAMINE, N-NITROSO-N-PROPYL-

SY - PROPYLAMINE, N-NITROSO-N-DI-

SY - RCRA WASTE NUMBER U111

CAREV-

- o CLASSIFICATION : B2; probable human carcinogen
- o BASIS FOR CLASSIFICATION : Increased tumor incidence at multiple sites in two rodent species and in monkeys administered the compound by various routes
- o HUMAN CARCINOGENICITY DATA :

Inadequate. Human exposure to nitrosamines results from contact with mixtures containing these compounds (e.g., cutting oils, tobacco products). Because of potential confounding by the other substances in these mixtures, data is of limited use in the evaluation of carcinogenicity of individual nitrosamines.

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o ANIMAL CARCINOGENICITY DATA :

Sufficient. As part of a survey of 65 N-nitroso compounds, Druckrey et al. (1967) administered N-nitrosodi-n-propylamine in drinking water to BD rats of unspecified sex. A total of 48 rats was treated in groups inferred to number 16, 16, 15 and 1 at doses of 4, 8, 15 or 30 mg/kg/day, respectively, for life. Of 48 treated animals, 45 developed liver carcinomas; tumor induction time was dose-related. Tumors of the esophagus and tongue were also observed.

N-nitrosodi-N-propylamine administered to male Sprague-Dawley rats in drinking water at 1.8 mg/day, 5 days/week for 30 weeks resulted in liver carcinomas (9/15), esophageal papillomas (6/15) and carcinomas (8/15) and nasal adenocarcinomas (8/15) (Lijinsky and Taylor, 1978, 1979). F344 rats of both sexes treated in a similar fashion with 0.9 mg/day developed esophageal carcinomas (20/20) and forestomach tumors (12/20) (Lijinsky and Reuber, 1981).

Corn oil gavage of male and female F344 rats (2 times/week for 30 weeks) produced nasal and liver carcinomas, and esophageal tumors; tumors at these sites were not found in controls (Lijinsky and Reuber, 1983).

A high incidence of malignant tumors at distant sites, primarily nasal cavity, liver and lungs, was observed in Sprague-Dawley rats of both genders receiving lifetime weekly s.c. injections of 24.36, 48.72 or 97.44 mg/kg N-nitrosodi-n-propylamine (Reznik et al., 1975). Similar studies in hamsters reported increases in tumors of the nasal cavities, laryngobronchial tract and lungs (Pour et al., 1973, Althoff et al., 1973).

Macaque monkeys given weekly i.p. injections of 40 mg N-nitrosodi-n-propylamine for a total dose of 70 g had a higher incidence of hepatocellular carcinomas (6/6) compared with that of presumed historical controls (7/90) (Adamson and Sieber, 1979, 1983).

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o SUPPORTING DATA :

N-nitrosodi-n-propylamine is mutagenic for *Salmonella typhimurium* (IARC, 1978; Phillipson and Ioannides, 1985), *E. coli* (McMahon et al., 1979; Probst et al., 1981; Rao et al., 1981) and V79 cells and mouse lymphoma cells (Kuroki et al., 1977; Bartsch et al., 1980; Jones and Huberman, 1980). Evidence of DNA damage by this compound includes unscheduled DNA synthesis in in vitro exposed rat hepatocytes and HeLa cells (Martin et al., 1978; Probst et al., 1981) DNA breakage in in vivo treated rat liver (Brambilla et al., 1981; Bradley et al., 1982) and chromosomal aberrations in Chinese hamster cells in vitro (Kaneko et al., 1978; Matsuka et al., 1979; Ishidate et al., 1981).

Both presumed and documented metabolites of N-nitrosodi-n-propylamine have been shown to be carcinogenic for hamsters and rats (IARC, 1978).

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CARO -

- |                               |  |
|-------------------------------|--|
| o CLASSIFICATION              | : B2; probable human carcinogen  |
| o BASIS FOR CLASSIFICATION    | : Increased tumor incidence at multiple sites in two rodent species and in monkeys administered the compound by various routes |
| o ORAL SLOPE FACTOR           | : $7.0E+0$ /mg/kg/day  |
| o DRINKING WATER UNIT RISK    | : $2.0E-4$ /ug/L   |
| o DOSE EXTRAPOLATION METHOD   | : One-hit  |
| o RISK/WATER CONCENTRATIONS : |  |

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	5E-1 ug/L
E-5 (1 in 100,000)	5E-2 ug/L
E-6 (1 in 1,000,000)	5E-3 ug/L

o ORAL DOSE-RESPONSE DATA :

Species/Strain Tumor Type	Dose Administered Human Equivalent	Tumor Incidence	Reference
Rat, BD, sex not specified; hepatocellular carcinomas	Route: Oral, drinking water		Druckrey, 1967; Druckrey et al., 1967

Information in the above references was used in quantitation of risk using the following relationship:

$Ck/(t50)^{**} n = d$

where: C = conversion between mmol and mg = 130.2 mg/mmol  
k = empirically derived constant estimated to be 1.7E+4 mmol/kg/day  
t50 = median time of tumor induction = 728  
n = representative value for dialkylnitrosamines as published by Druckrey = 2.3  
d = daily dose of test compound, calculated from the above to be 0.57831 mg/kg/day

The slope factor for rats (BA) was calculated from a rearrangement of the one-hit model:

$$BA = -\ln (0.5/\text{day}) = 1.20/\text{mg/kg/day}$$

Adjusting this value by the cube root of the assumed human body weight (70 kg) to the assumed rat body weight (0.35 kg) gives the human slope factor 7.02/mg/kg/day.

o ADDITIONAL COMMENTS :

A reported value of n=2.2 for N-nitrosodi-n-propylamine was not used since a k for this value was not reported. The k used was estimated from a plot of k vs number of C-atoms for lower di-n-alkylnitrosamines.

The unit risk should not be used if the water concentration exceeds 5E+1

ug/L, since above this concentration the slope factor may differ from that stated.

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o DISCUSSION OF CONFIDENCE :

Small numbers of rats were treated in groups of unspecified size. Sex of the animals was not reported nor were specific tumor incidences. There was no control group.

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CARDR-

o CARCINOGENICITY SOURCE :

U.S. EPA. 1986. Health and Environmental Effects Profile for Nitrosamines. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.

Druckrey, H. 1967. Quantitative aspects in chemical carcinogens. In: Potential Carcinogenic Hazards from Drugs. UICC Monograph, Series 7. Berlin Springer-Verlag. p. 60-78.

Druckrey, H., R. Pruessman, S. Ivankovic and D. Schmahl. 1967. Organotropic carcinogenic effect of 65 different N-nitroso compounds on BD rats. Z. Krebsforsch. 69: 103-201.

The 1986 Health and Environmental Effects Profile has received Agency review.

DOCUMENT

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o REVIEW DATES : 02/11/87  
o VERIFICATION DATE : 02/11/87  
o EPA CONTACTS :

James W. Holder / ORD -- (202)260-5721 / FTS 260-5721

Jim Cogliano / ORD -- (202)260-9243 / FTS 260-9243

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WQCHU-

Water and Fish Consumption: 8E-4 ug/L

Fish Consumption Only: 1.24E+0 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic effects because of exposure to diethylnitrosamine, the ambient water concentration should be zero. The criteria given represent an incremental risk of cancer over a lifetime of  $10^{-6}$ . The values are based on criteria for N-nitrosodiethylamine.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS  
(202)260-1315 / FTS 260-1315

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WQCAQ-

Freshwater:

Acute LEC --  $5.85E+3$  ug/L  
Chronic LEC -- none

Marine:

Acute LEC --  $3.3E+6$  ug/L  
Chronic LEC -- none

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LEC's are given when the minimum data required to derive water quality criteria are not available.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS  
(202)260-1315 / FTS 260-1315

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CERC -

Value -- 10 pounds (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The RQ for N-nitroso-N-propylamine is based on potential carcinogenicity. Available data indicate a hazard ranking of medium and a weight-of-evidence group B2, which corresponds to an RQ of 10 pounds.

Reference -- 54 FR 33418 (08/30/89)

EPA Contact -- RCRA/Superfund Hotline

(800) 424-9346 / (202) 260-3000 / FTS 260-3000

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RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)260-3000 / FTS 260-3000

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TSCA -

No data available

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OREF - None

IREF - None

CREF - Adamson, R.H. and S.M. Sieber. 1979. The use of nonhuman primates for chemical carcinogenesis studies. ISBN 0-12-192750-4.

CREF - Adamson, R.H. and S.M. Sieber. 1983. Chemical carcinogenesis studies in nonhuman primates. EPA-600/9-83-008. NTIS PB 83-220137.

CREF - Althoff, J., F.W. Krueger and U. Mohr. 1973. Brief communication: Carcinogenic effect of dipropylnitrosamine and compounds related by beta- oxidation. J. Natl. Cancer Inst. 51(1): 287-288.

CREF - Bartsch, H., C. Malaveille, A.M. Camus, et al. 1980. Validation and comparative studies on 180 chemicals with S. typhimurium strains and V79 Chinese hamster cells in the presence of various metabolizing systems. Mutat. Res. 76: 1-50.

CREF - Bradley, M.O., G. Dysart, K. Fitzsimmons, P. Harbach, J. Lewin and G. Wolf. 1982. Measurements by filter elution of DNA single- and double-strand breaks in rat hepatocytes: Effects of nitrosamines and gamma-irradiation. Cancer Res. 42(7): 2592-2597.

CREF - Brambilla, G., M. Cavanna, A. Pino and L. Robbiano. 1981. Quantitative correlation among DNA damaging potency of six N-nitroso compounds and their potency in inducing tumor growth and bacterial mutations. Carcinogenesis. 2(5): 425-429.

CREF - Druckrey, H., R. Preussmann, S. Ivankovic and D. Schmahl. 1967. Organotropism carcinogenic activities of 65 different N-Nitroso compounds in SD-rats. Z. Krebsforsch. 69(2): 103-201.

CREF - Druckrey, H. 1967. Quantitative aspects in chemical carcinogens. In: Potential Carcinogenic Hazards from Drugs, Evaluation of Risks, R. Truhart, Ed. UICC Monograph, Series 7. Berlin Springer-Verlag. p. 60-78.

CREF - IARC (International Agency for Research on Cancer). 1978. IARC



Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man N-Nitrosodimethylamine. Some N-Nitroso Compounds. WHO, IARC, Vol. 17, Lyon, France. p. 51, 77, 83, 125, 177, 221, 257.

- CREF - Ishidate, M., T. Sofuni and K. Yoshikawa. 1981. Chromosomal aberration tests in vitro as a primary screening tool for environmental mutagens and/or carcinogens. Gann Monogr. Cancer Res. 27: 95-108.
- CREF - Jones, C.A. and E. Huberman. 1980. A sensitive hepatocyte-mediated assay for the metabolism of nitrosamines to mutagens for mammalian cells. Cancer Res. 40(2): 406-411.
- CREF - Kaneko, A., M. Hayashi, K. Yoshikawa, et al. 1978. Chromosome aberration tests combined with S-9 metabolic activation system in vitro. Mutat. Res. 54: 240.
- CREF - Kuroki, T., C. Drevon and R. Montesano. 1977. Microsome-mediated mutagenesis in V79 Chinese hamster cells by various nitrosamines. Cancer Res. 37(4): 1044-1050.
- CREF - Lijinsky, W. and M.D. Reuber. 1981. Comparative carcinogenesis by some aliphatic nitrosamines in Fischer rats. Cancer Lett. 14(3): 297-302.
- CREF - Lijinsky, W. and M.D. Reuber. 1983. Carcinogenesis in Fischer rats by nitrosodipropylamine, nitrosodibutylamine and nitrosobis(2-oxopropyl)amine given by gavage. Cancer Lett. 19: 207-213.
- CREF - Lijinsky, W. and H.W. Taylor. 1979. Carcinogenicity of methylated derivatives of N-nitrosodiethylamine and related compounds in Sprague-Dawley rats. J. Natl. Cancer Inst. 62(2): 407-410.
- CREF - Lyjinsky, W. and H.W. Taylor. 1978. Comparative carcinogenicity of some derivatives of nitrosodi-n-propylamine in rats. Ecotoxicol. Environ. Saf. 2(3-4): 421-426.
- CREF - Martin, C.N., A.C. McDermid and R.C. Garner. 1978. Testing of known carcinogens and noncarcinogens for their ability to induce unscheduled DNA synthesis in HeLa cells. Cancer Res. 38(3): 2621-2627.
- CREF - Matsuoka, A., M. Hayashi and M. Ishidate, Jr. 1979. Chromosomal aberration tests on 29 chemicals combined with S9 mix in vitro. Mutat. Res. 66(3): 277-290.
- CREF - McMahon, R.E., J.C. Cline and C.Z. Thompson. 1979. Assay of 855 test chemicals in ten tester strains using a new modification of the ames test for bacterial mutagens. Cancer Res. 39(3): 682-693.
- CREF - Phillipson, C.E. and C. Ioannides. 1985. Metabolic activation of nitrosamines to mutagens by various animal species including man. Biochem. Pharmacol. 34(3): 441-442.
- CREF - Pour, P., F.W. Kruger, A. Cardesa, J. Althoff and U. Mohr. 1973. Carcinogenic effect of di-n-propylnitrosamine in Syrian golden hamsters. J. Natl. Cancer Inst. 51(3): 1019-1027.
- CREF - Probst, G.S., R.E. McMahon, L.E. Hill, C.Z. Thompson, J.K. Epp and S.B. Neal. 1981. Chemically-induced unscheduled DNA synthesis in primary rat hepatocyte cultures: A comparison with bacterial mutagenicity using 218 compounds. Environ. Mutagen. 3(1): 11-32.
- CREF - Rao, T.K., B.E. Allen, W. Winton, W. Lijinsky and J.L. Epler. 1981. Nitrosamine-induced mutagenesis in Escherichia coli K12 (343/113). 1. Mutagenic properties of certain aliphatic nitrosamines. Mutat. Res. 89(3): 209-215.
- CREF - Reznik, G., U. Mohr and F.W. Kruger. 1975. Carcinogenic effects of di-n-propylnitrosamine, beta-hydroxypropyl-n-propylnitrosamine and methyl-n-propylnitrosamine on Sprague-Dawley rats. J. Natl. Cancer Inst. 54(4): 937-943.
- CREF - U.S. EPA. 1986. Health and Environmental Effects Profile for

Nitrosamines. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.

HAREF- None

Option? TYPE 9/2

File 9; Entry 1; Accession No. 1459

(CAS) CAS Registry Number: 85-01-8

(MAT) Material Name: Phenanthrene

(SYN) Synonyms:

Phenanthrene;

HSDB 2166;

NSC 26256;

Phenanthren [German];

Phenanthrene

(UPD) Update Date: 12-01-90

(EFF) Effective Date: 07-01-91

(STAT) Status:

STATUS OF DATA FOR Phenanthrene

File On-Line 12-01-90

Category (section)	Status	Last
Revised		
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Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	
12-01-90		
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	no data	
Supplementary Data (V.)	no data	

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(CAR) Carcinogenicity Assessment:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D, not classifiable as to human carcinogenicity

Basis -- Based on no human data and inadequate data from a single gavage study in rats and skin painting and injection studies in mice.

II.A.2. HUMAN CARCINOGENICITY DATA

None.

II.A.3. ANIMAL CARCINOGENICITY DATA

Inadequate. Data from a rat gavage study and mouse skin application and injection studies are not adequate to assess the carcinogenicity of phenanthrene. Ten female Sprague-Dawley rats received a single oral dose of 200 mg phenanthrene in sesame oil (Huggins and Yang, 1962). No mammary tumors occurred. The observation period was not specified; however, based on the discussion of other experiments in the report it was probably at least 60 days. Controls were not reported.

Complete carcinogenic activity was not shown in two skin painting assays.

Kennaway (1924) reported no tumors in 100 mice (strain and sex not specified)

treated with phenanthrene (purity not specified) in 90% benzene (dose not reported) for 9 months. Roe and Grant (1964) reported in an abstract that

mice (number, sex and strain not specified) did not develop tumors after

dermal exposure to 5% phenanthrene (purity not specified, vehicle not specified) 3 times/week for 1 year.

Five studies of cancer-initiating activity in skin painting assays in mice

have yielded one positive result. Groups of 30 female CD-1 mice received a

single dermal application of 1.8 mg phenanthrene in benzene,

followed by  
twice-weekly applications of tetradecanoylphorbol acetate (TPA, 3 mg), a  
promoter, for 35 weeks (Scribner, 1973). Phenanthrene used in  
the study was  
purified by preparative thin-layer chromatography (TLC) and  
determined to be  
homogeneous on TLC. It is stated in the report that the dose of  
TPA was 3 mg  
(5 umol); however, it is not clear whether this refers to the  
twice weekly or  
total dose. Controls were treated with TPA (6 mg); it is not  
clear whether  
controls received benzene (vehicle). The tumor incidence (skin  
papilloma) at  
35 weeks was 12/30 (40%) in treated mice and 0/30 in TPA  
controls.

Tumor-initiating activity was not shown in the four other  
mouse skin  
painting studies. In the first study, male Swiss albino  
(Ha/ICR) mice (15 to  
20/group) received 10 applications of a 0.1% solution of  
phenanthrene in  
acetone (total dose 1 mg) or acetone alone, followed by repeated  
applications  
of TPA (2.5 ug in acetone) 3 times/week for 20 weeks (LaVoie et  
al., 1981).  
Phenanthrene was >99.5% pure as determined by high pressure  
liquid  
chromatography (HPLC). No tumors occurred in treated or control  
mice. Wood  
et al. (1979) exposed female CD-1 mice (30/group) to a single  
application of  
1.8 mg phenanthrene in acetone:ammonium hydroxide (1000:1) or  
vehicle alone,  
followed by TPA (10 ug) twice weekly for 35 weeks. Phenanthrene  
used in this  
study was >98% pure and homogeneous on HPLC. Tumor incidence  
(skin  
papillomas) out of 27-29 survivors in each group was 17% in  
treated mice and  
7% in vehicle controls (not statistically different). In  
another study,  
albino mice (10/sex/dose, strain not specified) received four  
dermal  
applications of phenanthrene (total dose 1.2 mg, purity not  
specified) in  
acetone or to acetone alone, followed by croton oil once each  
week for 20  
weeks (Roe, 1962). Tumor incidence (skin papillomas) was 4/19  
(21%) in  
treated mice and 2/20 (10%) in vehicle controls. In the last  
study (Salaman

and Roe, 1956), groups of 20 "S" strain mice (sex unspecified) received 10 dermal applications (3 times/week) of 18% phenanthrene (total dose 0.54 g, purity not specified) in acetone, followed by 18 weekly applications of croton oil. Controls were treated with 18 applications of croton oil; 10 controls survived until termination. The tumor incidence (skin papillomas) was 5/20 (25%) in treated mice and 4/10 (40%) in croton oil controls.

Parenterally administered phenanthrene was not shown to have tumorigenic activity in three studies. In the first (Buening et al., 1979), groups of Swiss Webster BLU:Ha ICR mice (100/group, approximately 50% of each sex) received intraperitoneal injections of phenanthrene (total dose 0.25 mg) in dimethyl sulfoxide (DMSO) or DMSO alone on days 1, 8, and 15 after birth. Phenanthrene was >98% pure and homogeneous on HPLC. Incidence of pulmonary

tumors (adenomas) at 38 to 42 weeks was 1/18 (6%) and 5/17 (30%) in female and male treated mice and 7/38 (18%) and 2/10 (19%) in female and male controls; the apparent differences were not statistically significant. No hepatic tumors occurred in treated or control mice. One treated female mouse developed malignant lymphoma. In the second study (Grant and Roe, 1963), albino mice (sex, strain and group size not specified) received single subcutaneous injections of phenanthrene (40 ug, purity not specified) in an acetone/gelatin vehicle or only the vehicle. Incidence of pulmonary adenomas after 52-62 weeks was 3/39 (6%) in treated mice and 8/34 (24%) in vehicle controls. Other tumors reported were 4 hepatomas and 2 skin papillomas in treated mice, and 1 mammary adenocarcinoma, 1 hepatoma and 1 hemangioma in control mice. Finally in the Steiner (1955) study, groups of 40 to 50 male and female C57BL mice (numbers per sex not specified) received single subcutaneous injections of 5 mg phenanthrene (purity not specified) in tricapylin. No tumors were reported in 27 surviving mice after

4 months.

Vehicle controls were not reported.

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Phenanthrene has not yielded positive results in assays for DNA damage in *Bacillus subtilis* and *Escherichia coli* (Rosenkrantz and Poirier, 1979; McCarroll et al., 1981). Tests for mutagenicity in *Salmonella typhimurium* have yielded positive (Oesch et al., 1981; Sakai et al., 1985; Bos et al., 1988) and negative results (Wood et al., 1979; McCann et al., 1975; LaVoie et al., 1981; Kaden et al., 1979; Bos et al., 1988). The results of phenanthrene in a fungi recombination assay (Simmon, 1979) and in tests for DNA damage in several mammalian cell cultures were not positive (Lake et al., 1978; Probst et al., 1981; Rice et al., 1984). A test for forward mutation in Chinese hamster ovary cells exposed to 1 ug/mL was not positive (Huberman and Sachs, 1976), whereas a test in human lymphoblast TK6 cells incubated with rat liver S9 (Arochlor) and 9 ug/mL phenanthrene yielded positive results (Barfknecht et al., 1981). Phenanthrene did not yield positive results in sister chromatid exchange and chromosome aberration assays in mammalian cell cultures (Popescu et al., 1977) or in cell transformation assays in several types of mammalian cells (5-40 ug/mL) (Marquardt and Heidelberger, 1972; Kakunaga, 1973; Evans and DiPaolo, 1975; Pienta et al., 1977).

Current theories regarding the mechanisms of metabolic activation of polycyclic aromatic hydrocarbons lead to predictions of a carcinogenic potential for phenanthrene. Jerina et al. (1978) considered phenanthrene to have a "bay-region" structure. It is metabolized by mixed function oxidases to reactive diol epoxides (Nordqvist et al., 1981; Vyas et al., 1982) that have been shown to be weakly mutagenic in some bacterial and mammalian cell assays (Wood et al., 1979). Evidence from in vivo assays indicates, however, that phenanthrene metabolites have a relatively low tumorigenic

potential.

The 1,2-, 3,4- and 9,10-dihydrodiol metabolites of phenanthrene did not show tumor initiating activity in mouse skin painting assays (Wood et al., 1979).

The 1,2-diol-3,4-epoxides of phenanthrene did not produce lung tumors when injected into newborn mice (Buening et al., 1979). The relatively weak mutagenic and tumorigenic activity of phenanthrene diol epoxides is

inconsistent with the "bay region theory" of PAH carcinogenesis. The reason

for the inconsistency has not been elucidated. Phenanthrene epoxides have a

relatively small molecular size (relative to other more active PAH epoxides

such as chrysene diol epoxides) and as a result may have a lower affinity for

DNA or may be transported less efficiently into the mammalian nucleus (Wood et

al., 1979). While some studies have considered phenanthrene to have a "bay-

region" structure, it may not clearly fall into this category.

## II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

None.

## II.C. QUANTITATIVE ESTIMATE OF RISK FROM INHALATION EXPOSURE

None.

## II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

The 1990 Drinking Water Criteria Document for Polycyclic Aromatic

Hydrocarbons (PAHs) has received Agency and external review.

### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1990. Drinking Water Criteria Document for Polycyclic Aromatic

Hydrocarbons (PAHs). Prepared by the Office of Health and Environmental

Assessment, Environmental Criteria and Assessment Office,



Cincinnati, OH for  
the Office of Drinking Water, Washington, DC. Final Draft.  
ECAO-CIN-D010,  
September, 1990.

II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The 1990 Drinking Water Criteria Document for Polycyclic  
Aromatic  
Hydrocarbons has received Agency and external review.

Agency Work Group Review: 02/07/90, 05/03/90

Verification Date: 05/03/90

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Rita Schoeny / ORD -- (513)569-7544 / FTS 684-7544

Robert McGaughy / ORD -- (202)382-5889 / FTS 382-5889

Option? TYPE 11/2

File 11; Entry 1; Accession No. 1445

(CAS) CAS Registry Number: 129-00-0

(MAT) Material Name: Pyrene

(SYN) Synonyms:  
BENZO(DEF)PHENANTHRENE;  
HSDB 4023;  
NSC 17534;  
PYREN [GERMAN];  
PYRENE;  
BETA-PYRENE

(UPD) Update Date: 07-01-91

(EFF) Effective Date: 07-01-91

(STAT) Status:

STATUS OF DATA FOR Pyrene

File On-Line 09-01-90

Category (section)	Status	Last
Revised		
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Oral RfD Assessment (I.A.) 07-01-91	on-line	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.) 01-01-91	on-line	
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	no data	
Supplementary Data (V.) 09-01-90	on-line	

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(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.A.1. ORAL RfD SUMMARY

Critical Effect RfD	Experimental Doses*	UF	MF
-----	-----	-----	---
Kidney effects (renal 3E-2 tubular pathology, mg/kg/day decreased kidney weights)	NOAEL: 75 mg/kg/day  LOAEL: 125 mg/kg/day	3000	1

Mouse Subchronic  
Oral Bioassay

U.S. EPA, 1989

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\*Conversion Factors: None

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

U.S. EPA. 1989. Mouse Oral Subchronic Toxicity of Pyrene.  
Study conducted  
by Toxicity Research Laboratories, Muskegon, MI for the Office  
of Solid Waste,

Washington, DC.

Male and female CD-1 mice (20/sex/group) were gavaged with  
0, 75, 125, or  
250 mg/kg/day pyrene in corn oil for 13 weeks. The  
toxicological parameters  
examined in this study included body weight changes, food  
consumption,  
mortality, clinical pathological evaluations of major organs and  
tissues, and  
hematology and serum chemistry. Nephropathy, characterized by  
the presence of  
multiple foci of renal tubular regeneration, often accompanied  
by interstitial  
lymphocytic infiltrates and/or foci of interstitial fibrosis,  
was present in  
4, 1, 1, and 9 male mice in the control, low-, medium-, and

high-dose groups,  
respectively. Similar lesions were seen in 2, 3, 7, and 10  
female mice in the  
0, 75, 125, and 250 mg/kg treatment groups. The kidney lesions  
were described  
as minimal or mild in all dose groups. Relative and absolute  
kidney weights  
were reduced in the two higher dosage groups. Based on the  
results of this  
study, the low dose (75 mg/kg/day) was considered the NOAEL and  
125 mg/kg/day  
the LOAEL for nephropathy and decreased kidney weights.

#### I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF = 3000. An uncertainty factor of 3000 reflects 10 each for  
intra- and  
interspecies variability, 10 for the use of a subchronic study  
for chronic RfD  
derivation, and an additional 3 to account for the lack of both  
toxicity  
studies in a second species and developmental/reproductive  
studies.

MF = 1.

#### I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

White and White (1939) fed six male rats (unspecified  
strain) a diet  
containing 2000 mg pyrene/kg for 40 days. The average reported  
food intake  
for two animals was 6.1 g/day, and the average body weight for  
these two  
animals was 94.3 g. A decrease in body weight gain was observed  
in two  
animals. The authors stated that this body weight gain was  
representative of  
the whole group; although there was no change in food intake.  
White and White  
(1939) also observed enlarged livers and increased hepatic lipid  
content in  
animals treated with pyrene, benzpyrene or methylcholanthrene in  
the diet;  
however, incidence data were not reported and it is unclear  
whether this  
effect occurred in the pyrene treated rats. Interpretation of  
this study is  
further complicated by the lack of experimental controls and  
statistical  
analysis, small sample size, and incomplete reporting of  
histopathology  
results.

I.A.5. CONFIDENCE IN THE ORAL RfD

Study: Medium  
Data Base: Low  
RfD: Low

Confidence in the principal study is medium, as it was a well-designed experiment that examined a variety of toxicological endpoints and identified both a NOAEL and LOAEL for the critical effect. Confidence in the data base is low, due to the lack of supporting subchronic, chronic, and developmental/reproductive studies. Accordingly, confidence in the RfD is low.

I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- This assessment is not presented in any existing U.S. EPA document.

Agency Work Group Review: 11/15/89

Verification Date: 11/15/89

I.A.7. EPA CONTACTS (ORAL RfD)

Harlal Choudhury / ORD -- (513)569-7536 / FTS 684-7536

Kenneth A. Poirier / ORD -- (513)569-7553 / FTS 684-7553

(CAR) Carcinogenicity Assessment:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity

Basis -- Based on no human data and inadequate data from animal bioassays.

II.A.2. HUMAN CARCINOGENICITY DATA

None.

II.A.3. ANIMAL CARCINOGENICITY DATA

Inadequate. Groups of 14-29 newborn male and 18-49 newborn female CD-1 mice on 1, 8, and 15 days of age received intraperitoneal injections of pyrene (purity unknown) in dimethyl sulfoxide (DMSO) (total dose = 40, 141 or 466 ug/mouse), or DMSO alone (Wislocki et al., 1986). Tumors were evaluated in animals that died spontaneously after weaning and in all remaining animals at 1 year after exposure. The mid-dose group was initiated 10 weeks after the other groups and had a separate vehicle control. The survival rate in the high-dose groups (male and female) was 25 to 35%; most of the mice died between the last injection and weaning. This high mortality was not observed in the control, low- or mid-dose groups (the survival rates were not stated). A statistically significant increase in the incidence of liver carcinomas occurred in the mid-dose males (3/25) relative to their vehicle control group (0/45), but not in the high-dose males (1/14) or low-dose males (0/29) or in female mice, when compared with their respective controls. The incidences of total liver tumors (adenomas and carcinomas), lung tumors or malignant lymphomas were not statistically significantly elevated in treated animals. The results of this 1-year experiment were not considered to be positive because of the overall lack of tumorigenic response in the short-term.

Mouse skin-painting assays of pyrene as a complete skin carcinogen or as an initiator of carcinogenicity were either not positive or inconclusive (Badger et al., 1940; Horton and Christian, 1974; Van Duuren and Goldschmidt, 1976; Salaman and Roe, 1956; Scribner, 1973).

A subcutaneous pyrene injection did not produce tumors in Jackson A mice; the mice were observed for 18 months after injection (Shear and Leiter, 1941).

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

In DNA damage assays in *Escherichia coli* and *Bacillus*

subtilis pyrene was not mutagenic (Ashby and Kilbey, 1981). In bacterial gene mutation tests both positive (Kinae et al., 1981; Bridges et al., 1981; Matijasevic and Zeiger, 1985; Sakai et al., 1985; Kaden et al., 1979; Bos et al., 1988) and negative (McCann et al., 1975; LaVoie et al., 1979; Ho et al., 1981; Bos et al., 1988) results have been reported. The consensus conclusion on the international collaborative study (which involved 20 bacterial test sets) was that protocol or evaluation criteria were critical factors in individual test verdicts. Pyrene induced increased incidence of mitotic gene conversion but not other genetic endpoints in yeast (de Serres and Hoffman, 1981). Pyrene did not induce an increase in sex-linked recessive lethals in *Drosophila* (Valencia and Houtchens, 1981).

Mixed results have also been observed in mammalian assays in vitro, again with protocol and evaluation criteria being a factor in at least some of the cases. In the collaborative study Evans and Mitchell (1981) concluded pyrene was positive for SCE induction in CHO cells when all concentrations were different from controls, but no apparent increase when the concentration was increased 10-fold. In the same volume, two other laboratories reported pyrene negative both for SCE and for chromosome aberrations in CHO cells (Brookes and Preston, 1981). Tong et al. (1981) also reported that pyrene did not induce SCE in a rat liver epithelial cell system. Jotz and Mitchell (1981) reported pyrene was positive in the L5178Y mouse lymphoma gene mutation assay.

Pyrene did not induce chromosome aberrations (as detected by micronuclei) or SCE in bone marrow of several mouse strains receiving i.p. injections of pyrene (Purchase and Ray, 1981). Results of mammalian cell transformation assays in a variety of cell types have not been positive (DiPaolo et al., 1969; Pienta et al., 1977; Casto, 1979; Chen and Heidelberger,

1969; DiPaolo  
et al., 1972; Kakunaga, 1973; Evans and DiPaolo, 1975).

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM SOIL  
EXPOSURE

None.

II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM  
INHALATION EXPOSURE

None.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS  
(CARCINOGENICITY ASSESSMENT)

II.D.1. EPA DOCUMENTATION

U.S. EPA. 1990. Drinking Water Criteria Document for  
Polycyclic Aromatic  
Hydrocarbons (PAHs). Prepared by the Office of Health and  
Environmental  
Assessment, Environmental Criteria and Assessment Office,  
Cincinnati, OH for  
the Office of Drinking Water, Washington, DC. ECAO-CIN-D010,  
September, 1990.  
(Final Draft)

II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The 1990 Drinking Water Criteria Document for Polycyclic  
Aromatic  
Hydrocarbons has undergone Agency and external review.

Agency Work Group Review: 02/07/90

Verification Date: 02/07/90

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Rita S. Schoeny / ORD -- (513)569-7544 / FTS 684-7544

Robert E. McGaughy / ORD -- (202)382-5889 / FTS 382-5889

(PROP) Physical-Chemical Properties:

V.B. PHYSICAL-CHEMICAL PROPERTIES



Chemical Formula: C16H10

Molecular Weight: 202.26

Boiling Point: 759F, 404C (Merck, 1976) (SUSPECT)

Specific Gravity (H2O=1): 1.27 at 23C (Merck, 1976)

Vapor Pressure (mmHg): Not Found

Melting Point: 313F, 156C (Merck, 1976)

Vapor Density (AIR=1): Not Found

Evaporation Rate (Butyl acetate=1): Not Found

Solubility in Water: 0.135 mg/liter in water (MacKay, 1977)

Appearance and Odor: Colorless solid (Sax, 1984, p. 2324);  
solid and  
solutions have a slight blue fluorescence (Merck, 1983, p. 1149)

Flash Point [Method Used]: Not Found

Flammable Limits -- Not Found

Conditions or Materials to Avoid -- Not Found

Hazardous Decomposition or Byproducts -- When heated to  
decomposition, pyrene  
emits acrid smoke and fumes (Sax, 1984, p. 2324).

Use -- Biochemical research (Hawley, 1981, p. 872).

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Option? TYPE 14/2

File 14; Entry 1; Accession No. 1472

(CAS) CAS Registry Number: 7782-49-2

(MAT) Material Name: Selenium and Compounds

(SYN) Synonyms:

Selenium;  
C.I. 77805;  
Caswell No. 732;  
ELEMENTAL SELENIUM;  
EPA Pesticide Chemical Code 072001;  
HSDB 4493;  
SELEN [Polish];  
Selenio [Spanish];  
Selenium;  
SELENIUM ALLOY;  
SELENIUM BASE;  
SELENIUM DUST;  
SELENIUM ELEMENTAL;  
SELENIUM HOMOPOLYMER;  
UN 2658;  
Selenic acid, disodium salt;  
Caswell No. 791;  
Disodium selenate;  
EPA Pesticide Chemical Code 072002;  
Natriumseleniat [German];  
NSC 378348;  
Selenic acid, disodium salt;  
Sodium selenate;  
Selenious acid, disodium salt;  
DISODIUM SELENITE;  
DISODIUM SELENIUM TRIOXIDE;  
HSDB 768;  
Natriumselenit [German];  
SELENIOUS ACID, DISODIUM SALT;  
SODIUM SELENITE;  
UN 2630;  
Selenious acid;  
HSDB 6065;  
MONOHYDRATED SELENIUM DIOXIDE;  
Selenious Acid;  
Selenic acid;  
Acide selenique [French];  
Acido selenico [Spanish];  
HSDB 675;  
Selenic acid;  
UN 1905;

Sodium selenide [Na<sub>2</sub>Se];  
Disodium monoselenide;  
Sodium selenide

(UPD) Update Date: 06-01-91

(EFF) Effective Date: 07-01-91

(STAT) Status:  
STATUS OF DATA FOR Selenium and Compounds

File On-Line 03-01-91

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	06-01-91
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	06-01-91
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	no data	
Supplementary Data (V.)	no data	

(HAZ) Chronic Health Hazards, Noncarcinogenic:  
I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS  
I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)  
I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Clinical selenosis	NOAEL: 0.015 mg/kg/day	3	1	5E-3

mg/kg/day

Human Epidemiological Study LOAEL: 0.023 mg/kg/day

Yang et al., 1989b

\*Conversion Factors: NOAEL (0.853 mg/day) and LOAEL (1.261 mg/day) calculated from regression analysis ( $\log Y = 0.767 \log X - 2.248$ , where Y = blood selenium and X = selenium intake) as detailed in Yang et al. (1989a) based

upon the correlation ( $r = 0.962$ ) between dietary selenium intake and blood selenium level for data showing incidence of clinical selenosis in adults based on an average adult body weight of 55 kg (Yang et al., 1989b).

#### I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Yang, G., S. Yin, R. Zhou, et al. 1989b. Studies of safe maximal daily dietary Se-intake in a seleniferous area in China. II. Relation between Se-intake and the manifestation of clinical signs and certain biochemical alterations in blood and urine. J. Trace Elem. Electrolytes Health Dis. 3(2): 123-130.

Yang et al. (1989b), in a follow-up to an earlier study (Yang et al., 1983), studied a population of approximately 400 individuals living in an area of China with unusually high environmental concentrations of selenium (Se).

The subjects were evaluated for clinical and biochemical signs of Se intoxication. Three geographical areas with low, medium and high selenium levels in the soil and food supply were chosen for comparison in the studies.

The earlier Yang et al. (1983) study was conducted in response to endemic selenium intoxication in two separate areas with sample sizes of only 6 and 3. Comparisons were then made to a selenium-adequate area ( $n=8$ ) and low-selenium area ( $n=13$ ). The Yang et al. (1989a,b) studies provide a much larger sample size and include additional analysis of tissue selenium levels. This allows a more accurate estimation of the dose-response relationship observed for selenium toxicity. Selenium levels in soil and approximately 30 typical food types commonly eaten by the exposed population showed a positive correlation with blood and tissue Se levels. The daily average Se intakes, based on lifetime exposure, 70, 195 and 1438 ug for adult males and 62, 198 and 1238 ug for adult females in the low-, medium- and high-selenium areas, respectively.

Significant correlations demonstrated between Se concentrations of various tissues were used to estimate the minimal daily Se intake values that elicited various alterations in biochemical parameters indicative of possible Se-induced liver dysfunction (i.e., prolongation of clotting time and serum glutathione titer) and clinical signs of selenosis (i.e., hair or nail loss, morphological changes of the nails, etc.). In this manner, a marginal safe level of daily Se intake was estimated.

Analysis of the results indicated that persistent clinical signs of

selenosis were observed only in 5/439 adults, a potentially sensitive subpopulation. The blood selenium concentration in this group ranged from 1.054 to 1.854 mg/L with a mean of 1.346 mg/L. Clinical signs observed included the characteristic "garlic odor" of excess selenium excretion in the breath and urine, thickened and brittle nails, hair and nail loss, lowered hemoglobin levels, mottled teeth, skin lesions and CNS abnormalities (peripheral anesthesia, acroparesthesia and pain in the extremities). Alterations in the measured biochemical parameters occurred at dietary intake levels of 750-850 ug/day. These alterations were described as a delay in prothrombin time, i.e., increase in blood coagulation time and reduction in blood glutathione concentration. However, these indicators were poorly characterized and are not typically used as an index for clinical selenosis resulting from chronic exposure to selenium (NAS, 1989). Based upon the blood selenium levels shown to reflect clinical signs of selenium intoxication, a whole blood selenium concentration of 1.35 mg/L corresponding to 1.261 mg of daily selenium intake is indicative of the lowest correlative selenium intake causing overt signs of selenosis. The next lowest whole blood selenium concentration of 1.0 mg/L, corresponding to 0.853 mg selenium/day, produces no clinical signs of selenosis. The NOAEL for this study is 0.85 mg Se/day and the LOAEL is 1.26 mg Se/day.

A group of 142 volunteers in South Dakota and Wyoming were recruited by Longnecker et al. (1991) at random from households listed in a telephone directory or from ranches with suspected high selenium intake based on previous cases of livestock selenosis. The geographical areas were chosen because of known seleniferous topsoil and high concentrations of selenium in plants and food. The subjects were followed for 1 year and completed health questionnaires, underwent physical examinations, provided blood samples for clinical assessment, and provided blood, urine, toenails and duplicate-plate food collections for selenium analysis. The average selenium intake was 239 ug/day, approximately 2-3 times higher than the national average. The concentration of selenium in whole blood, serum, urine and toenails and the amount in diet were highly correlated. Blood selenium concentration was highly correlated with selenium intake. The correlation was very similar to that reported by Yang et al. (1989a). Liver function (prothrombin time and

alanine aminotransferase, aspartate aminotransferase, gamma glutamyltransferase and alkaline phosphatase), hematologic function (leukocyte count, hemoglobin and hematocrit) and clinical chemistry (sodium, potassium and chloride concentration) were not found to be altered as a result of selenium intake. High regression coefficient predictor variables for selenium toxicity (muscle twitching, paresthesia, nail loss, nail lines, hair loss and garlic breath) were not found in increased frequency for this population. No signs of selenium toxicity were found in this population, including individuals whose selenium intake was as high as 724 ug/day. This report corroborates that of Yang et al. (1989b), which showed that a selenium intake of up to 853 ug/day is not associated with characteristic nail or hair loss typical of selenium intoxication.

#### I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF = 3. An uncertainty factor of 3 was applied to the NOAEL to account for sensitive individuals. A full factor of 10 was not deemed necessary since a moderately-sized human population was exposed to high levels of selenium throughout a lifetime, the essential requirement for selenium, and because of the purported beneficial anticarcinogenic attributes of excess selenium in the diet.

MF = 1.

#### I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

The essentiality for selenium has been well-documented in livestock based upon the alleviation of specific deficiency conditions by selenium supplementation of the diet (Combs and Combs, 1986). Selenium has been clearly demonstrated to be a cofactor of glutathione peroxidase, a hydroperoxide and lipid peroxide reducing enzyme and is therefore essential (Rotruck et al., 1973). Human requirements for selenium were not conclusively established until 1979 when an association was made between low selenium status and cardiomyopathy (Keshan disease) in China for young children and women of child-bearing age (Keshan Disease Research Group, 1979a,b). More recently, iatrogenic episodes of selenium deficiency have been reported in patients receiving intravenous total parenteral administration of feeding solutions devoid of selenium. Symptoms included low glutathione peroxidase activity and low selenium levels in erythrocytes (Lavander and Burk, 1986), muscular

weakness and discomfort (van Rij et al., 1979) and cardiomyopathy (Johnson et al., 1981). It is important to note that glutathione peroxidase activity is a valid indicator of human selenium status only in populations with relatively low selenium intakes, since the enzyme activity plateaus at adequate selenium intake levels (Whanger et al., 1988), thereby precluding the use of this biochemical indicator under excessive selenium intake situations.

The NAS (1989) has determined the recommended dietary allowance for selenium to be 0.87 ug/kg, or approximately 70 and 55 ug/day for the reference adult North American male and female, respectively. Requirements for selenium increase during pregnancy to 65 ug/day and for lactation to 75 ug/day.

Selenium requirements for infants and children vary according to age. However, based on the reference weights of NHANES II, these populations demonstrate an increased requirement per unit weight relative to adults. For

infants, the selenium requirement is 1.67 ug/kg and for children the requirement ranges from 1.07-1.53 ug/kg. It should be noted that the most

recent RDA for selenium did not consider the 1989 results of Yang et al. (1989a,b) discussed above, but an earlier preliminary report by the same authors (Yang et al., 1983).

Yang et al. (1983) reported clinical signs of selenosis (i.e., loss of hair and nails) in approximately 50% of a population of 248 inhabitants living in Enshi County, Hubei Province of the People's Republic of China. Selenosis was reported in the highest selenium contaminated area where the average daily Se intake was 5.0 mg/day (range 3.2-6.7), but no selenosis occurred when the average intake was 0.750 mg/day (range 0.240-1.51). These estimates, however, were based upon estimates of intake from only 6 and 3 inhabitants in the high and low contaminated areas, respectively. Yang et al. (1989b) reported prolonged clotting time and serum glutathione and these biochemical changes were indicated as adverse effects of selenium exposure. Glutathione is a strong nucleophile that reacts well with soft electrophiles and is an important conjugate-forming compound for the detoxification and excretion of electrophilic metabolites and metabolically produced oxidizing agents. If glutathione is depleted or markedly reduced in the liver, the hepatotoxicity of these compounds would likewise be expected to be enhanced (Ketterer et al., 1983). However, the significance of decreased serum glutathione is not well characterized and should not be used in this context as a biochemical marker

of selenium toxicity. Likewise, there is no indication that prothrombin activity is affected by excess selenium administration (Longnecker et al., 1991). Furthermore, the description of this effect in Yang et al. (1989b) was based on a population for which there is insufficient documentation of normal clotting times in the general Chinese population.

Selenium toxicity has been clinically described according to three types: acute selenosis, subacute selenosis and chronic selenosis. The acute condition is caused by consuming relatively high amounts of selenium over a short period of time. After the onset of this condition, walking becomes unsteady, cyanosis of the mucous membranes occurs and labored breathing is usually seen sometimes resulting in death. Pathological findings include congestion of the liver, endocarditis and myocarditis, degeneration of the smooth musculature of the gastrointestinal tract, gallbladder and bladder, and erosion of the long bones (Francke and Moxon, 1936).

Subacute selenosis occurs from exposure to large doses of Se over a longer period of time resulting in neurological dysfunction (impaired vision, ataxia, disorientation) and respiratory distress. It is typically seen most frequently in grazing livestock feeding upon Se-accumulating plants and has been referred to as "blind staggers" (Rosenfeld and Beath, 1964).

Prolonged exposure to more moderate levels of selenium result in skin lesions involving alopecia, hoof necrosis and loss, emaciation and increased serum transaminases and alkaline phosphatase in animals. In man, the condition is characterized by chronic dermatitis, fatigue, anorexia, gastroenteritis, hepatic degeneration, enlarged spleen and increased concentrations of Se in the hair and nails (Harr and Muth, 1972).

Selenium exists naturally in a number of oxidation states, thereby accounting for the different forms of selenium important to living organisms by oral ingestion. In the -2 oxidation state, selenium can be found as hydrogen selenide ( $H_2Se$ ), sodium selenide ( $Na_2Se$ ), di- $[(CH_3)_2Se]$  and trimethyl selenium  $[(CH_3)_3Se]$  and various selenoamino acids such as selenomethionine, selenocysteine, Se-methyl selenocysteine, selenocystathionine and selenotaurine. Elemental selenium and the dipeptide selenodiglutathione have an oxidation state of 0. In the +4 oxidation state, selenium can exist as selenium dioxide ( $SeO_2$ ), selenious acid ( $H_2SeO_3$ ) or as sodium selenite ( $Na_2SeO_3$ ). Finally, in its most oxidized state (+6), selenium can be found as



selenic acid ( $\text{H}_2\text{SeO}_4$ ) or as sodium selenate ( $\text{Na}_2\text{SeO}_4$ ).

The toxicity of selenium has been consistently well documented. However, some early studies reported that selenium may be a carcinogen. Nelson et al. (1943) showed that rats fed diets containing Se as seleniferous wheat developed hepatic tumors and low-grade carcinomas in 11/53 animals. This work has subsequently been criticized due to low-protein content and relatively high levels of Se in the diet (5, 7 or 10 ppm Se), a poorly characterized source of selenium, and in general poor experimental design. The authors reported no encapsulation or metastases and in fact noted their own difficulty in determining the difference between hyperplasia and tumor. Another early investigation by Seifter et al. (1946) reported several thyroid tumors and adenomatous hyperplasia in livers of rats fed 0.05% bis-4-acetylaminophenyl selenium dihydroxide for 105 days. This organic selenium compound was suspected of having goitrogenic properties but its carcinogenic effect has not been further confirmed to be attributable to the selenium in the molecule.

The first animal experiment which demonstrated anticarcinogenic effects of selenium was performed by Clayton and Baumann (1949). An approximate 50% reduction in dimethylaminoazobenzene-induced tumor incidence occurred in rats fed a diet supplemented with 5 ppm Se as selenite. Additional evidence subsequently reported, further illustrated the inhibitory effect of selenium on transplantable tumors in rats (Weisberger and Suhrland, 1956a) and leukemia in humans (Weisberger and Suhrland, 1956b). The National Cancer Institute sponsored an extensive study on selenium toxicity in rats in order to resolve the issue of selenium carcinogenicity. Diets containing up to 8 ppm selenium did not increase tumor incidence (Tinsley et al., 1967; Harr et al., 1967). Since 1970, there has been an increased interest in characterizing the anti-carcinogenic and anti-tumorigenic properties of selenium. The number of reports characterizing these properties are too numerous to discuss in detail here. The reader is referred to a review by Milner and Fico (1987) for a more comprehensive treatment of the data base.

The essentiality and toxicity of selenium varies according to the valence

state of selenium when incorporated into biomolecules and the form in which selenium is fed or administered. This is especially true when comparing the LD50 value as an index of toxicity for the various selenium compounds. Although it is difficult to make an assessment for several selenium compounds:

by a similar mode of administration in a common species, there is general agreement that sodium selenite, sodium selenate, selenomethionine and selenogluthathione are among the more toxic species (Combs and Combs, 1986).

The relative potency of systemic toxicity for selenium compounds is also similar in experiments examining potency of anti-tumorigenic activity. In

vitro examination of potency of effect of selenium compounds on incubated Ehrlich ascites tumor cells (EATC) showed that sodium selenite is more efficacious in significantly reducing EATC viability than an equivalent concentration of sodium selenate. Although selenium dioxide, selenomethionine and selenocystine ultimately decreased viability of the EATC, nearly 50% more

incubation time was required for the same effect (Poirier and Milner, 1979).

The same authors investigated the relative potency of various selenium compounds administered intraperitoneally on EATC growth in vivo. Sodium selenite and selenodigluthathione (an intermediate of selenium metabolism) were the most effective forms of selenium in preventing EATC propagation. Sodium

selenide, dimethyl selenide and selenocystine were not effective in inhibiting EATC growth (Poirier and Milner, 1983). Similar relative potency results have been reported in in vitro systems for canine mammary cells (Fico et al., 1986) and human mammary cells (Watrach et al., 1984).

Since selenium has been reported to cause growth retardation, decreased fertility, embryotoxicity, fetotoxicity and teratogenic effects in animals,

Yang et al. (1989b) made the following observations: Malformation in chickens hatched from locally produced eggs did occur; however, teratogenic effects in human infants were never seen in this area although Se has been reported to be transmitted through the placenta to the fetus in animals. These findings confirm those reported by Yang et al. (1983) in which chicken eggs from this

same area were reported to have very low hatchability and some deformed embryos in those that did hatch.

The developmental toxicity of selenomethionine was investigated by Tarantal et al. (1991) in non-human primates. Forty pregnant long-tailed macaques were dosed daily by nasogastric intubation with 0, 0.025, 0.150 or 0.3 mg selenium/kg as selenomethionine through gestational days 20-50. Dams were examined clinically and the pregnancies of two to three dams within each

test group were followed to term (gestational day 165). All other dams were hysterectomized on gestational day 100. Neonates delivered at term were examined for morphometric, neurologic, behavioral and ophthalmologic effects on days 1, 8, 15, 22 and 30. Pregnancy loss among treated animals was not significantly different from concurrent or historical controls. No statistically significant treatment-related effects were observed at necropsy on gestational day 100. There were no significant maternal or fetal developmental effects or teratogenesis found up to 0.3 mg/kg selenium, the highest dose tested.

Halverson et al. (1966) fed 60-70 g male, post-weanling Sprague-Dawley rats selenium as selenite or seleniferous wheat ad libitum at 1.6, 3.2, 4.8, 6.4, 8.0, 9.6 or 11.2 ppm of selenium (13, 27, 40, 67, 81 or 94 ug/kg/day, respectively). Levels of selenium up to 4.8 ppm showed no effect. At 8.0 ppm selenium as seleniferous wheat, there was an observed decrease in liver weight, increase in spleen weight, and decrease in hemoglobin. Mortality was observed in the groups fed 8.0, 9.6 and 11.2 ppm selenium as seleniferous wheat at incidences of 1/8, 5/8 and 8/8, respectively. The incidences of mortality reported for groups fed 8.0 and 9.6 ppm selenium as selenite were 1/8 and 1/10, respectively. A significant growth reduction was reported for both selenium sources at 6.4 ppm and higher, although feed utilization was not decreased. No other effects were reported for the rats fed sodium selenite.

Schroeder and Mitchener (1971) administered 3 ppm selenium as selenate (390 ug/kg/day) to CD mice through four generations. Maternal effects were not observed. There was a significant increase in young deaths in the F1 generation and an increase in numbers of runts in generations F1 through F3. By the F3 generation there was also a decrease in breeding events.

Rosenfeld and Beath (1954) administered selenium as potassium selenate to sires and pregnant rats through five breeding cycles at 1.5, 2.5 or 7.5 ppm selenium (75, 125 or 375 ug/kg/day). No effect was observed on reproduction, the number of young reared or on the reproduction of two successive generations of dams and sires in groups receiving 1.5 ppm selenium. In the

group receiving 2.5 ppm selenium, there was a 50% reduction in the number of young reared. At 7.5 ppm there was a decrease in fertility of the females but not males, a decrease in the number of survivors and a reduction in the rate of growth in the young.

Nobunaga et al. (1979) administered 3 or 6 ppm selenium (390 or 780 ug/kg/day, respectively) as selenite to IVCS mice for 30 days prior to mating and throughout gestation. On day 18 of gestation, maternal mice were sacrificed and the embryos removed. Number of litters, total implants, total implants per dam, dead fetuses, dead embryos, resorptions, surviving fetuses (% to total implants), litter size, gross malformations and skeletal anomalies were not significantly different for either selenium-treated or control mice. The only significant effect noted was a decrease in the body weight of surviving fetuses in mice given 6 ppm selenium.

#### I.A.5. CONFIDENCE IN THE ORAL RfD

Study: Medium  
Data Base: High  
RfD: Medium

Confidence in the chosen principal study is medium. Although this is a human epidemiological study in which a sizable population with sensitive subpopulations was studied, there are still several possible interactions that were not fully accounted for, e.g., fluoride intake and protein status. Also, except for clinical signs of selenosis there are no other reliable indicators, biochemical or clinical, of selenium toxicity. Confidence in the data base is high because many animal studies and epidemiologic studies (reviewed by Combs and Combs, 1986) support the principal study. Medium to high confidence in the RfD is selected based upon the critical study and RfD levels of confidence.

#### I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation -- U.S. EPA, 1985

Agency Work Group Review: 01/20/88, 03/22/89, 09/21/89, 11/14/90, 03/27/91

Verification Date: 03/27/91

I.A.7. EPA CONTACTS (ORAL RfD)

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(CAR) Carcinogenicity Assessment:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

NOTE: This assessment is for the following compounds: Selenium (CASRN 7782-49-2); sodium selenate (CASRN 13410-01-0); sodium selenite (CASRN 10102-18-8); selenious acid (CASRN 7783-00-8); selenic acid (CASRN 7783-08-6); sodium selenide (CASRN 1313-85-5).

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to carcinogenicity in humans

Basis -- Based on inadequate human data and inadequate evidence of carcinogenicity in animals. The evidence for various selenium compounds in animal and mutagenicity studies is conflicting and difficult to interpret; however, evidence for selenium sulfide is sufficient for a B2 (probable human carcinogen) classification.

II.A.2. HUMAN CARCINOGENICITY DATA

Inadequate. Data on the potential carcinogenicity of selenium and various selenium compounds in humans are inadequate. Epidemiological studies have evaluated selenium in blood and cancer death rates in areas of high vs. low naturally-occurring selenium. However, these studies have limited value because they do not assess specific selenium compounds or correlate exposure with cancer risk.

Several investigators have studied the association between serum selenium and the risk of cancer through prospective, case-control and nested case-control studies. Analysis of blood serum levels indicated that patients with cancer, particularly gastrointestinal cancer, prostatic cancer, or Hodgkin's

lymphoma, had significantly lower blood selenium levels in blood than healthy patients (Shamberger et al., 1973; Salonen et al., 1984; Kok et al., 1987; Willet et al., 1983; Willet and Stampfer, 1986). The risk of cancer for men (Kok et al., 1987) or for all subjects (Willet et al., 1983) in the lowest quintile of serum selenium was twice that of subjects with higher levels.

Geographic correlational studies have compared cancer mortality in areas of high vs. low levels of naturally-occurring selenium. In an ecological study Shamberger and Frost (1969) reported that an inverse relationship existed between cancer death rates and the selenium concentrations in foliage plants of several Canadian provinces. The human cancer death rate in provinces with selenium-containing plants was  $122.2 \pm 7.8$  (presumably per 100,000 population although this was not specified), while in the provinces devoid of these plants, the human death rate was  $139.9 \pm 4.0$ .

In an ecological study Shamberger and Willis (1971) reported that there was a correlation between decreased cancer death rates in humans and an increase in the selenium in the forage crops in California. In high-selenium areas (selenium 0.11 ppm of forage crops) the cancer death rate per 100,000 was 141.2. In the medium-selenium areas (0.05-0.10 ppm) the cancer death rate was 190.1. In low-selenium areas (0.02-0.05 ppm) the cancer death rate was 233.0. Shamberger and Willis (1971) also investigated the ratio of observed to expected cancer death rates by anatomic site for men in 17 paired cities including high- and low-selenium areas. The anatomic sites that would come into contact with dietary selenium, such as pharynx, esophagus, stomach, bladder and intestine, showed a substantially lower rate ratio in the high-selenium cities than in the low-selenium cities. Other ecological and prospective studies have correlated an increased incidence of colon, breast and other forms of cancer in humans in geographic areas where selenium is deficient and a lowered cancer incidence with higher selenium concentrations (Schrauzer and Ishmael, 1974; Shamberger, 1976; Schrauzer et al., 1976; Jansson et al., 1978; Yang et al., 1983).

In a study of approximately 300 employees exposed to selenium (form not

specified) in a rectifier (electronics) process over a 26-year period, only 17 deaths occurred, 6 of which were because of cancer (Glover, 1970). This number, however, is not statistically different from the 5.1 deaths expected based on national mortality rates. The source of the mortality rates was not specified. Several toxic effects including pulmonary irritation, epigastric pain and dermal irritation and dermatitis were associated with selenium exposure in men, but no carcinogenic effect was reported.

#### II.A.3. ANIMAL CARCINOGENICITY DATA

Inadequate. The carcinogenicity of selenium compounds has been evaluated in several animal studies. However, the data are conflicting and difficult to interpret because of apparent anticarcinogenic activity and high toxicity of some selenium salts. In addition, comparison of the available data is difficult because several different salts with varying degrees of bioavailability were used in the assays.

In a 2-year dietary study reported by Nelson et al. (1943), Osborne-Mendel rats (sex not specified) were fed selenium in the form of seleniferous corn or wheat or ammonium potassium selenide at 5-10 ppm. Survival was lower in the treated rats; 53/126 (42%) rats fed selenium survived 18 months or longer compared with 14/18 (78%) control rats. Of the 53 surviving selenium-treated rats, 43 (81%) developed liver cirrhosis and 11 (21%) developed hepatocellular adenoma or carcinoma. All 11 animals with tumors also had liver cirrhosis. None of the 14 control animals surviving 2 years developed liver tumors. Only pooled group data were reported and no statistical analysis was reported.

No tumors developed in a total of 1437 Wistar rats fed sodium selenite or sodium selenate in the diet at levels of 0.5-16 ppm for their lifetime (Harr et al., 1967; Tinsley et al., 1967). Nonneoplastic liver effects such as hyperemia, cellular degeneration, binucleation, and mild proliferation of hepatocytes were observed at concentrations of 4 ppm and higher.

Long-Evans rats (approximately 50/sex/group at study initiation) received 2 ppm (as selenium) sodium selenate or sodium selenite in drinking water for 1 year, then 3 ppm for the remainder of the study (Schroeder and Mitchener, 1971). The treatment of the control group was not discussed. The animals were observed for the duration of their natural lifespan, approximately 36 months, although one selenate-treated female lived for 5 years. Selenite

produced 50% mortality in males by 58 days. At this time, 2 ppm selenate was substituted for selenite in the male group. The concentration of selenium was raised to 3 ppm in this group when the animals were 1 year old; however, the high mortality rendered the group size too small for further statistical analysis. Selenite produced 50% mortality in females by 348 days; selenite-treated females were sacrificed at 23 months due to high mortality. Selenate produced 50% mortality in females by 1014 days and in males by 962 days. In the control groups 50% mortality was achieved by 872 and 853 days in females and males, respectively. Survival of rats receiving selenate was comparable to controls and median lifespan was increased by >100 days. Body weights of treated males were comparable to controls throughout the study. Body weights of females fed selenate were significantly greater than controls at 24 and 36 months; body weights of females fed selenite were significantly less than controls at all times but 18 months.

Incidence of all tumors and of malignant tumors was significantly increased in the selenate-treated rats compared with the controls. Incidence of all tumors in controls, selenate- and selenite-treated rats was 20/65 (30.8%), 30/48 (62.5%) and 4/32 (12.5%), respectively. Incidence of malignant tumors in the same groups was 11/65 (16.9%), 20/48 (41.7%) and 4/32 (12.5%), respectively. The earliest tumor occurred on day 833 in the control males, on day 633 in the control females, on day 344 in selenate males and on day 633 in selenate females. The shortened survival time of the selenite groups was thought to be responsible for the small number of tumors. This study is considered inadequate because only the heart, lung, liver, kidney and spleen tissues from animals necropsied were examined histologically, and an increase in longevity was observed in selenate-treated female rats.

Schroeder and Mitchener (1972) administered 3 ppm sodium selenate or sodium selenite in drinking water to Swiss mice (50/sex/group). Body weights of selenate-treated animals were comparable to controls. Body weights of males fed selenite were significantly increased compared with controls, but body weights of females fed selenite were significantly decreased compared with controls. Longevity in males fed selenate was increased compared with controls. Longevity in females fed selenate increased, but longevity in



females fed selenite decreased compared with controls. When compared to controls, there was no significant increase in total tumor incidence or malignant tumor incidence observed in selenium- (form not specified) treated mice. In the control group 23/119 (19%) had tumors (10/119 (8%) malignant tumors). Selenium-fed mice showed 13/88 (15%) tumors (all were malignant). In selenium-treated group 8/13 malignancies were lymphoma or leukemia, 4/13 were papillary or alveologenic adenocarcinoma and 1/13 an osteosarcoma. In the control group there were two incidences of lymphoma or leukemia, 7 of lung carcinoma and 1 carcinoma of unknown origin. The 13 benign tumors included breast and ovary tumors.

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Selenium is an essential micronutrient for several species, including humans, and is part of several enzymes such as glutathione peroxidase, an enzyme involved in cellular defense against oxidative damage, and heme oxidase. While low doses of selenium are essential, high doses of selenium or a deficiency of dietary selenium may cause a toxic response. Additionally, selenium may be protective against tumor development. The greatest daily exposure to selenium is via food. Bioavailability of selenium is dependent on numerous factors, including the intake levels, chemical form and nutritional status. Organic forms of selenium are more bioavailable than inorganic forms; selenates and selenites are the inorganic forms more readily absorbed. Sodium selenate and selenite are soluble in water, but the extent to which they are absorbed dermally or through the gastrointestinal tract has not been fully elucidated (U.S. EPA, 1989).

Shamberger (1985) reported that the oral administration of 0.1-6 ppm or dermal application of 0.005% of selenium reduced incidences of skin, liver, tracheal, intestinal and lung tumors induced by several carcinogens in rats, mice and hamsters. Shamberger theorized that selenium may reduce cellular damage caused by peroxidation of fat. In another study, natural killer (NK) cell activity was significantly increased in female rats administered 0.5 or 2.0 ppm selenium (sodium selenate) in the drinking water for 10 weeks (Koller et al., 1986), suggesting to the authors that NK-sensitive tumors may be prevented by using selenium therapy.

Data on the mutagenicity of selenium and its compounds are equivocal. Selenate and selenite (12 uM) were mutagenic in a reverse mutation assay using *Salmonella typhimurium* strains TA98, TA100 and TA1537 (Noda et al., 1979) in the absence of rat hepatic homogenates. In a second assay, sodium selenate, but not sodium selenite, was mutagenic; the *S. typhimurium* strains used were not reported (Lofroth and Ames, 1978). Selenite (selenious acid and sodium selenite) produced DNA damage in *Bacillus subtilis* strains 17A and 45T; however, selenate (selenic acid and sodium selenate) was negative in the Rec assay (Nakamuro et al., 1976).

Sodium selenide, sodium selenite, and sodium selenate (in order of decreasing activity) caused an increase in unscheduled DNA synthesis in the presence or absence of glutathione in Chinese hamster ovary cells at concentrations of  $1.0 \times 10^{-4}$  M (Whiting et al., 1980). Increased chromosomal aberrations were also produced by sodium selenite at  $5 \times 10^{-5}$  M in rat lymphocytes (Newton and Lilly, 1986) and by sodium selenite, selenious acid, selenic acid, and selenium oxide at  $2.6 \times 10^{-6}$  M in human lymphocytes (Nakamuro et al., 1976). Sodium selenite produced an increase in chromosomal aberrations in the bone marrow of rats administered a total of 10-12 mg/kg intravenously (near-lethal doses) (Newton and Lilly, 1986). Selenium (elemental), selenium dioxide, sodium selenide, and sodium selenite (in order of decreasing activity) induced an increase in SCEs in human whole-blood cultures; sodium selenate was not mutagenic in this assay (Ray and Altenburg, 1980).

#### II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

None.

#### II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

None.

#### II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

#### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1980. Ambient Water Quality Criteria for Selenium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Quality Planning and Standards, Washington, DC. EPA 440/5-80-070. NTIS PB 81-117814.

U.S. EPA. 1984. Health Effects Assessment for Selenium (and Compounds). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Emergency and Remedial Response, Washington, DC. EPA/540/1-86-058. NTIS PB 86-134699.

U.S. EPA. 1989. Health and Environmental Effects Document for Selenium and Compounds. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.

#### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The 1989 Health and Environmental Effects Document on Selenium and Compounds has received OHEA review.

Agency Work Group Review: 11/09/89, 03/07/90

Verification Date: 03/07/90

#### II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

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Option? TYPE 15/2

File 15; Entry 1; Accession No. 1099

(CAS) CAS Registry Number: 7440-22-4

(MAT) Material Name: Silver

(SYN) Synonyms:  
ARGENTUM CREDE;  
COLLARGOL;  
Silver

(UPD) Update Date: 03-01-91

(EFF) Effective Date: 07-01-91

(STAT) Status:  
STATUS OF DATA FOR Silver

File On-Line 01-31-87

Category (section)	Status	Last Revised
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Oral RfD Assessment (I.A.)	on-line	03-01-91
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	06-01-89
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	on-line	03-01-88
Supplementary Data (V.)	no data	

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(HAZ) Chronic Health Hazards, Noncarcinogenic:  
I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS  
I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

NOTE: The Oral RfD for Silver may change in the near future pending the outcome of a further review now being conducted by the RfD/RfC Work Group.

#### I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
-----	-----	-----	---	-----
Argyria	NOAEL: None	2	1	3E-3 mg/kg/day
1-3 Year Therapeutic Treatments in Humans				
Gaul and Staud, 1935	LOAEL: 1.0 g (total i.v. dose)			
Blumberg and Carey, 1934	LOAEL: 6.4 g (total oral dose)			
East et al., 1980	LOAEL: 7.2 g (total oral dose estimated)			
	Average dose = 0.0052 mg/kg/day			
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#### \*Conversion Factors:

1000 mg x 1/0.18 x 1/70 kg x 1/25,500 days = 0.0031 mg/kg day;  
 6400 mg/32.7 kg/25,500 days = 0.0077 mg/kg/day;  
 7200 mg/58.6 kg/25,500 days = 0.0048 mg/kg/day;  
 Average = 0.0052 mg/kg/day

#### I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Gaul, L.E. and A.N. Staud. 1935. Clinical spectroscopy. Seventy cases of generalized argyria following organic and colloidal silver medication. J. Am. Med. Assoc. 104: 1387-1390.

Blumberg, H. and T.N. Carey. 1934. Argiremia: Detection of unsuspected and obscure argyria by the spectrographic demonstration of high blood silver. J. Am. Med. Assoc. 103: 1521-1524.

East, B.W., K. Boddy, E.D. Williams, D. MacIntyre and A.L.C. McLay. 1980.

Silver retention, total body silver and tissue silver concentrations in argyria associated with exposure to an anti-smoking remedy containing silver acetate. Clin. Exp. Dermatol. 5: 305-311.

In Gaul and Staud (1935), the LOAEL of 1.0 g was representative of the

lowest total doses (0.9-1.5 g) of silver associated with argyria in humans.

The doses were administered i.v. over a 2- to 3-year period as silver arsphenamine. No body weight data were reported.

Blumberg and Carey (1934) estimated the total dose from a dosing schedule for silver nitrate taken orally for 1 year as 6.4 g. The subject was an emaciated adult female (32.7 kg).

East et al. (1980) estimated the total body content of silver in one individual with argyria to be 6.4 (plus or minus 2) g. The subject ingested an unknown quantity of silver acetate over a period of 2.5 years. Symptoms of argyria appeared after the first 6 months of exposure. This subject retained 18% of a single dose of orally-administered silver in a separate 30-week experiment. Body weight was given as 58.6 kg.

Argyria is considered adverse beyond its cosmetic effect since it is irreversible and can be clinically mistaken for cyanosis.

Total dose is the most appropriate parameter because argyria is a cumulative effect of silver. The i.v. to oral conversion factor of 1/0.18 is based on the East et al. (1980) retention study. Pharmacokinetic studies in animals suggest that this value (18%) is high and should be considered a conservative estimate. Human body weight defaults to 70 kg in the absence of reported values. The total body burden of silver reported in East et al. (1980) was adjusted for the time of onset of argyria and then converted to an oral dose in the following manner:

$$6.4 \text{ g} \times 6 \text{ months}/30 \text{ months} = 1.3 \text{ g body burden}; 1.3 \text{ g}/0.18 = 7.2 \text{ g}.$$

#### I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF = 2. The standard UF of 10 for the intraspecies (human) variability is not considered appropriate because the affected subjects are of generally poor health and are considered to be sensitive elements of the population.

A UF of 2 is used for the LOAEL because the critical effect is considered to be minimally severe.

MF = 1

#### I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

The supporting animal data suggest that the RfD based on the human data should not be lower.

I.A.5. CONFIDENCE IN THE ORAL RfD

Study: Medium  
Data Base: Medium  
RfD: Medium

The human studies rate a medium confidence; they are reasonably good, with some quantitative dosing data. The data base confidence is medium because the existing animal studies quantitatively support the RfD. Medium confidence in the RfD follows.

I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

U.S. EPA. 1985. Drinking Water Criteria Document for Silver. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. (External Review Draft)

The 1985 Office of Drinking Water document has received Agency review and has been reviewed by several outside experts.

Agency Work Group Review: 10/09/85, 02/05/86

Verification Date: 10/09/85

I.A.7. EPA CONTACTS (ORAL RfD)

Julie Du / ODW -- (202)382-7583 / FTS 382-7583

Michael L. Dourson / ORD -- (513)569-7573 / FTS 684-7573

(CAR) Carcinogenicity Assessment:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classified as to human carcinogenicity

Basis -- In animals, local sarcomas have been induced after implantation of foils and discs of silver. However, the interpretation of these findings has

been questioned due to the phenomenon of solid-state carcinogenesis in which even insoluble solids such as plastic have been shown to result in local fibrosarcomas.

#### II.A.2. HUMAN CARCINOGENICITY DATA

No evidence of cancer in humans has been reported despite frequent therapeutic use of the compound over the years.

#### II.A.3. ANIMAL CARCINOGENICITY DATA

Inadequate. Local sarcomas have been induced after subcutaneous (s.c.) implantation of foils and discs of silver and other noble metals. Furst (1979, 1981), however, cited studies showing that even insoluble solids such as smooth ivory and plastic result in local fibrosarcomas and that tin when crumbled will not. He concluded that i.p. and s.c. implants are invalid as indicators of carcinogenicity because a phenomenon called solid-state carcinogenesis may complicate the interpretation of the cause of these tumors. It is difficult to interpret these implantation site tumors in laboratory animals in terms of exposure to humans via ingestion. Within these constraints there are two studies given below in which silver per se appeared to induce no carcinogenic response.

Schmahl and Steinhoff (1960) reported, in a study of silver and of gold, that colloidal silver injected both i.v. and s.c. into rats resulted in tumors in 8 of 26 rats which survived longer than 14 months. In 6 of the 8, the tumor was at the site of the s.c. injection. In about 700 untreated rats the rate of spontaneous tumor formation of any site was 1 to 3%. No vehicle control was reported.

Furst and Schlauder (1977) evaluated silver and gold for carcinogenicity in a study designed to avoid solid-state carcinogenesis. Metal powder was suspended in trioctanoin and injected monthly, i.m., into 50 male and female Fischer 344 rats per group. The dose was 5 mg each for 5 treatments and 10 mg each for 5 more treatments for a total dose of 75 mg silver. The treatment regimen included a vehicle control (a reportedly inert material), and cadmium as a positive control. Injection site sarcomas were found only in vehicle control (1/50), gold (1/50) and cadmium (30/50); no tumors (0/50) appeared at the site of injection in the silver-treated animals. A complete necropsy was



performed on all animals. The authors mentioned the existence of spontaneous tumors in Fischer 344 rats, but reported only injection site tumors. They concluded that finely divided silver powder injected i.m. does not induce cancer.

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Further support for the lack of silver's ability to induce or promote cancer stems from the finding that, despite long standing and frequent therapeutic usage in humans, there are no reports of cancer associated with silver. In a recent Proceedings of a Workshop/Conference on the Role of Metals in Carcinogenesis (1981) containing 24 articles on animal bioassays, epidemiology, biochemistry, mutagenicity, and enhancement and inhibition of carcinogenesis, silver was not included as a metal of carcinogenic concern.

No evidence of the mutagenicity of silver was shown in two available studies. Demerec et al. (1951) studied silver nitrate for the possible induction of back-mutations from streptomycin dependence to nondependence in *Escherichia coli*. Silver nitrate was considered nonmutagenic in this assay. Nishioka (1975) screened silver chloride with other chemicals for mutagenic effects using a method called the rec-assay. Silver chloride was considered nonmutagenic in this assay.

#### II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

##### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1988. Drinking Water Criteria Document for Silver. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. ECAO-CIN-026. Final Draft.

##### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The 1988 Drinking Water Criteria Document for Silver has received Agency review.

Agency Work Group Review: 09/22/88

Verification Date: 09/22/88

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

W. Bruce Peirano / ORD -- (513)569-7540 / FTS 684-7540

Julie Du / ODW -- (202)382-7583 / FTS 382-7583

(REGS) Regulations:

III. HEALTH HAZARD ASSESSMENTS FOR VARIED EXPOSURE DURATIONS

IV. U.S. EPA REGULATORY ACTIONS

IV.A. CLEAN AIR ACT (CAA)

No data available

IV.B. SAFE DRINKING WATER ACT (SDWA)

IV.B.1. MAXIMUM CONTAMINANT LEVEL (MCL) for Drinking Water

Value (status) -- 0.05 mg/L (1980)

Considers technological or economic feasibility? -- NO

Discussion --

Reference -- 45 FR 57332 (08/27/80)

EPA Contact -- James Murphy / Criteria and Standards Division, ODW /  
(202)382-7571 / FTS 382-7571; or Drinking Water Hotline / (800)426-4791

IV.C. CLEAN WATER ACT (CWA)

IV.C.1. AMBIENT WATER QUALITY CRITERIA, Human Health

Water and Fish Consumption: 5E+1 ug/L

Fish Consumption Only: None

Considers technological or economic feasibility? -- NO

Discussion -- This value is the same as the drinking water standard and approximates a safe level assuming consumption of contaminated organisms and water.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division, OWRS  
(202)475-7315 / FTS 475-7315

#### IV.C.2. AMBIENT WATER QUALITY CRITERIA, Aquatic Organisms

##### Freshwater:

Acute -- Varies with hardness  
Chronic LEC --  $1.2E-1$  ug/L

##### Marine:

Acute --  $2.3E+0$  ug/L  
Chronic -- None

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LECs are given when the minimum data required to derive water quality criteria are not available. The freshwater acute criterion varies with water hardness. For freshwater aquatic life the concentration (in ug/L) of total recoverable silver should not exceed the numerical value given by the equation " $e^{(1.72 [\ln (\text{hardness})] - 6.52)}$ "

(\*\* indicates exponentiation; hardness is in mg/L). For example, at a hardness of 50 mg/L, the acute WQC would be 1.2 and, at a hardness of 100 mg/L, the criterion would be 4.1 mg/L.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division, OWRS  
(202)475-7315 / FTS 475-7315

#### IV.F. RESOURCE CONSERVATION AND RECOVERY ACT (RCRA)

##### IV.F.1. RCRA APPENDIX IX, for Ground Water Monitoring

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

#### IV.G. SUPERFUND (CERCLA)

##### IV.G.1. REPORTABLE QUANTITY (RQ) for Release into the Environment

Value (status) -- 1000 pounds (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ is based on chronic toxicity. RQ assignments based on chronic toxicity reflect two primary attributes of the hazardous substance, the minimum effective dose (MED) levels for chronic exposure (mg/day for 70 kg person) and the type of effect (liver necrosis, teratogenicity, etc). A composite score is determined from an evaluation of these two attributes. Silver was determined to have a composite score of between 6 and 20, corresponding to a chronic toxicity RQ of 1000 pounds.

Reference -- 50 FR 13456 (04/04/85)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

Captured 8/12/92

1 - IRIS  
IRSN - 115  
DATE - 920807  
UPDT - 08/07/92, 52 fields  
STAT - Oral Rfd Assessment (RDO) on-line 08/01/90  
STAT - Inhalation RfC Assessment (RDI) on-line 08/01/92  
STAT - Carcinogenicity Assessment (CAR) on-line 08/01/90  
STAT - Drinking Water Health Advisories (DWHA) on-line 09/01/90  
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 04/01/92  
IRH - 03/01/88 RDO Text revised  
IRH - 09/07/88 CAR Carcinogen summary on-line  
IRH - 02/01/89 CARDR Secondary contact's phone number corrected  
IRH - 07/01/89 RDI Inhalation Rfd now under review  
IRH - 03/01/90 REFS Bibliography on-line  
IRH - 04/01/90 CREF Combs et al., 1973 citation corrected  
IRH - 06/01/90 CAA Area code for EPA contact corrected  
IRH - 06/01/90 RCRA EPA contact changed  
IRH - 07/01/90 RDO Withdrawn; new Rfd verified (in preparation)  
IRH - 07/01/90 OREF Oral Rfd references withdrawn  
IRH - 08/01/90 RDO Oral Rfd summary replaced; Rfd changed  
IRH - 08/01/90 CAR Text edited  
IRH - 08/01/90 OREF Oral Rfd references revised  
IRH - 09/01/90 HADV Health Advisory on-line  
IRH - 09/01/90 HAREF Health Advisory references added  
IRH - 08/01/91 CREF Litton Bionetics, Inc., 1981 reference title clarified  
IRH - 01/01/92 EXSR Regulatory actions updated  
IRH - 04/01/92 CAA CAA regulatory action withdrawn  
IRH - 08/01/92 RDI Inhalation RfC on-line  
IRH - 08/01/92 IREF Inhalation references on-line  
RLEN - 71690  
NAME - Toluene  
RN - 108-88-3  
SY - ANTISAL 1a  
SY - BENZENE, METHYL  
SY - METHACIDE  
SY - METHYL-BENZENE  
SY - METHYLBENZOL  
SY - NCI-C07272  
SY - PHENYL-METHANE  
SY - RCRA WASTE NUMBER U220  
SY - TOLUEEN  
SY - TOLUEN  
SY - Toluene  
SY - TOLUOL  
SY - TOLUOLO  
SY - TOLU-SOL  
SY - UN 1294

RDO -

o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	NF	RFD
Changes in liver and kidney weights	NOAEL: 312 mg/kg converted to 223 mg/kg/day	1000	1	2E-1 mg/kg/day
13-Week Rat Gavage Study	LOAEL: 625 mg/kg converted to 446 mg/kg/day			

\*Conversion Factors: Dose adjusted for gavage schedule of 5 days/week.

o ORAL RFD STUDIES :

NTP (National Toxicology Program). 1989. Toxicology and Carcinogenesis Studies of toluene in F344/N rats and B6C3F1 mice. Technical Report Series

No. 371. Research Triangle Park, NC.

The oral toxicity of toluene was investigated in this subchronic gavage study in F344 rats. Groups of 10 rats/sex/group were administered toluene in corn oil at dosage levels of 0, 312, 625, 1250, 2500, or 5000 mg/kg for 5 days/week for 13 weeks. All animals receiving 5000 mg/kg died within the first week. One female and 8 males in the 2500 mg/kg group died, but 2 of these were due to gavage errors. No deaths occurred at lower doses. Several toxic effects were noted at doses greater than or equal to 2500 mg/kg, including prostration, hypoactivity, ataxia, piloerection, lacrimation, excessive salivation, and body tremors. No signs of biologic significance were seen in groups receiving less than or equal to 1250 mg/kg. The only significant change in body weight was a decrease ( $p < 0.05$ ) for males in the 2500 mg/kg group. There were no toxicologically significant changes in hematology or urinalysis for any group of animals. Biochemical changes, including a significant increase ( $p < 0.05$ ) in SGOT in 2500 males and a dose-related increase in cholinesterase in females receiving 2500 and 5000 mg/kg, were not considered to be biologically significant. There were several pathologic findings and organ weight changes in the liver, kidney, brain, and urinary bladder. In males, absolute and relative weights of both the liver and kidney were significantly increased ( $p < 0.05$ ) at doses greater than or equal to 625 mg/kg. In females, absolute and relative weights of the liver, kidney, and heart were all significantly increased at doses greater than or equal to 1250 mg/kg ( $p < 0.01$  for all comparisons except  $p < 0.05$  for absolute kidney and heart weights at 1250 mg/kg). Histopathologic lesions in the liver consisted of hepatocellular hypertrophy, occurring at greater than or equal to 2500 mg/kg. Nephrosis was observed in rats that died, and damage to the tubular epithelia of the kidney occurred in terminally sacrificed rats. Histopathologic changes were also noted in the brain and urinary bladder. In the brain, mineralized foci and necrosis of neuronal cells were observed in males and females at 2500 mg/kg and males at 1250 mg/kg. In the bladder, hemorrhage of the muscularis was seen in males and females at 5000 mg/kg and males at 2500 mg/kg. The NOAEL for this study is 312 mg/kg/day based on liver and kidney weight changes in male rats at 625 mg/kg. The toxicologic significance of these organ weight changes is strengthened by the occurrence of histopathologic changes in both the liver and kidney at higher doses. Because the exposure was for 5 days/week, this dose is converted to  $312 \times 5/7 = 223$  mg/kg/day. The LOAEL is 625 mg/kg, which is 446 mg/kg/day when converted.

NTP (1989) also conducted a 13-week gavage study in B6C3F1 mice, following the same regimen described above. All mice receiving 5000 mg/kg died and 8/20 receiving 2500 mg/kg also died. Signs of toxicity seen in animals receiving greater than or equal to 2500 mg/kg included subconvulsive jerking, prostration, impaired grasping reflex, bradypnea, hypothermia, ataxia, and hypoactivity. By week 13, the mean body weight of 2500 mg/kg males was significantly ( $p < 0.05$ ) lower than controls. No other significant changes were reported for any group, including macroscopic observation, organ weight means, or clinical pathology parameters. The NOAEL for mice in this study was 1250 mg/kg.

The subchronic study by Wolf et al. (1956) is supportive of the NTP studies. Groups of 10 female Wistar rats were administered gavage doses of 0, 118, 354, or 590 mg/kg toluene dissolved in olive oil. A total of 138 doses were administered over 193 days, resulting in average doses of approximately 0, 84, 253, or 422 mg/kg/day. Hematologic, behavioral, gross and histopathologic examinations were conducted with no toxic effects being reported at any dose. Therefore, the highest dose of 422 mg/kg/day is considered to be the NOAEL for this study. However, this study is not used as the basis for the RfD because the LOAEL of 446 mg/kg/day identified by NTP (1989) is too close to the NOAEL identified by Wolf et al. (1956). Also, the NTP study indicated that male rats are more sensitive to toluene and the Wolf study utilized only female rats.

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o ORAL RFD UNCERTAINTY :

UF = 1000. An uncertainty factor of 1000 was applied to account for inter- and intraspecies extrapolations, for subchronic-to-chronic extrapolation and for limited reproductive and developmental toxicity data.  
.....

o ORAL RFD MODIFYING FACTOR :

MF = 1.

o ORAL RFD COMMENTS :

Kostas and Hotchin (1981) exposed NYLAR mice pre- and post-natally to toluene provided in the drinking water at concentrations of 0, 16, 80, or 400 ppm. Effects were noted in all dosed groups on rotorod performance, measured at 45 to 55 days of age, but there was an inverse dose-response relationship. No effects of toluene exposure were seen on maternal fluid consumption, offspring mortality rate, development of eye or ear openings, or surface-righting response. This study is not suitable for use in risk assessment because only 6 to 9 pregnancies/dose group were obtained, and because the dose-response relationship was inverse.

In an abstract providing limited information, Nawrot and Staples (1979) reported an increase in embryonic lethality in mice exposed to toluene from days 6 to 15 of gestation. Pregnant CD-1 dams were administered 0.3, 0.5, or 1.0 mL/kg bw, 3 times/day (equivalent to approximate trichlorotrifluoroethane at either 500 mg/cu.m levels for 11 years or 5358 mg/cu.m levels for 2.77 years (Imbus and Adkins, 1972).

Slight impairment of psychomotor performance was reported in male volunteers exposed to trichlorotrifluoroethane concentrations of 19,161 mg/cu.m

for 2.75 hours (Stopps and McLaughlin, 1967). This exposure period was too brief to consider a NOAEL for chronic exposure. Therefore, the RfD of 30 mg/kg/day is consi NTP, 1989. The studies identify the following potential target organs: kidney (male rat); hematologic effects (mice); central nervous system (rats, mice, primates); developmental toxicity (rats, rabbits). It is beyond the scope of this oral RfD summary sheet to describe each of these studies, but the two chronic (2 year) inhalation studies are summarized briefly below.

In a 2-year inhalation study by NTP (1989), F344 rats (60/sex/group) were exposed to 0, 600, or 1200 ppm toluene and B6C3F1 mice (60/sex/group) to 0, 120, 600, or 1200 ppm toluene for 6.5 hours/day, 5 days/week. Ten animals/group (except male mice) were removed at 15 months for toxicologic evaluation. At 15 months, there was an increased incidence and severity of nonneoplastic lesions of the nasal cavity of exposed rats. Minimal hyperplasia of the bronchial epithelium was seen in 4/10 female mice at 1200 ppm. There were no significant differences in survival among any group of animals during the 2-year study. Mean body weights were generally similar for all groups throughout the study. Nephropathy was seen in almost all rats with the severity somewhat increased in exposed rats. There were also effects on the olfactory and respiratory epithelia of exposed rats. No biologically important lesions were seen in any groups of mice. There was no evidence of carcinogenicity for any group of animals in this study.

A chronic inhalation study in rats performed by CIIT (1980) failed to produce an adverse effect. Groups of 40 F344 rats/sex were exposed to 30, 100, or 300 ppm toluene for 6 hours/day, 5 days/week for 24 months. An unexposed group of 120 rats/sex served as a control. Clinical chemistry, hematology, and urinalysis testing were conducted at 18 and 24 months. All parameters measured at the termination of the study were normal except for a dose-related reduction in hematocrit values in females exposed to 100 and 300 ppm toluene. The highest dose of 300 ppm was considered to be a NOAEL.

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o ORAL RFD CONFIDENCE :

Study: High  
Data Base: Medium  
RfD: Medium

Confidence in the principal study is high because a sufficient number of animals/sex were tested in each of six dose groups (including vehicle controls) and many parameters were studied. The same protocol was tested in both mice and rats, with rats being identified as the more sensitive species. The data base is rated medium because it is supported by a 6-month oral study. It is not higher than medium because there is no reproductive study. Also,



the oral studies are all subchronic, with the critical study being only 13 weeks in duration. Medium confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

Source Document -- This assessment is not presented in any existing U.S. EPA document.

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o REVIEW DATES : 05/20/85, 08/05/85, 08/05/86, 05/17/90,  
06/20/90

o VERIFICATION DATE : 06/20/90

o EPA CONTACTS :

Sue Velazquez / ORD -- (513)569-7571

Krishan Khanna / OST -- (202)260-7588

-----  
RDI -

o INHALATION RFD STUDIES :

Foo, S., J. Jeyaratnam and D. Koh. 1990. Chronic neurobehavioral effects of toluene. Br. J. Ind. Med. 47(7): 480-484.

NTP (National Toxicology Program). 1990. Toxicology and carcinogenesis studies of toluene in F344/N rats and B6C3F1 mice (inhalation studies). NTP-TR-371. 253 p.

In humans, toluene is a known respiratory irritant with central nervous system (CNS) effects. Because available studies could not provide subthreshold (NOAEL) concentrations for either of these effects, the LOAELs for both effects need to be considered in developing the RfC. Consequently, the study of Foo et al. (1990) was used for the CNS effects, and that of the National Toxicology Program (NTP, 1990) for the irritant effects. Because the CNS effect was judged to be a more severe and relevant endpoint, the LOAEL for this effect was used for deriving the RfC. Further, this effect is supported by a number of other occupational studies that show effects around 100 ppm.

Foo et al. (1990) conducted a cross-sectional study involving 30 exposed female workers employed at an electronic assembly plant where toluene was emitted from glue. Toluene levels reported in the study were from personal sample monitoring and reported as an 8-hour TWA, although the number of samples taken and the actual sampling period were not given. No historical exposure values were given. Co-exposure to other solvents was not addressed in the study. The exposed and control cohorts were matched for age, ethnicity, and use of medications. Members of these cohorts did not use alcohol and were nonsmokers. Medical histories were taken to eliminate any histories of central or peripheral nervous system disorders. The average number of years (+/- SD) worked by the exposed population was 5.7 +/- 3.2 and by the controls was 2.5 +/- 2.7. Exposed workers breathed toluene air levels of 88 ppm (332 mg/cu.m) as a TWA and control workers 13 ppm (49 mg/cu.m) (TWA); both of which are averages of the individual personal samples. A battery of eight neurobehavioral tests were administered to all exposed and control workers. The tests were performed midweek, before the workers reported to their stations for the day. Group means revealed statistically significant differences in 6/8 tests; all tests showed that the exposed workers performed poorly compared with the control cohort. When individual test results were linearly regressed against personal exposure concentrations, poor concentration-response relationships resulted for the six tests, with correlation coefficients ranging from 0.44 to 0.30. Irritation effects were not evaluated in this study, and no clinical signs or symptoms were reported. The paucity of exposure information, coupled with the small size of the cohort, limits the interpretation of this study, although the results were essentially confirmed in a clinical study in which the toluene concentrations were carefully controlled (Echeverria et al., 1989) at levels bracketing 88 ppm. Although the data in Echeverria et al. (1989) were generated from short-

term exposures (3-7 hours over a period of 142 days), the results may be considered relevant to longer-term exposures as several studies indicate the absence of a duration-response relationship in toluene-induced symptomatology. Fornazzari et al. (1983) noted the absence of a duration-effect relationship among toluene abusers when they were segregated into neurologically impaired vs. unimpaired ( $p = 0.65$ ). The human studies of Iregren (1982), Cherry et al. (1985), Baelum et al. (1985), and the principal study of Foo et al. (1990) all report this lack of a duration-response relationship and confirm the occurrence of CNS effects. Foo et al. (1990) indicate a LOAEL of 88 ppm toluene (332 mg/cu.m) for neurobehavioral changes from chronic exposure to toluene.

In a 2-year bioassay, Fischer 344 rats (60/sex/group) were exposed to 0, 600, or 1200 ppm (0, 2261, or 4523 mg/cu.m, respectively) toluene vapors, 6.5 hours/day, 5 days/week (duration-adjusted to 0, 437, and 875 mg/cu.m, respectively) for 103 weeks (NTP, 1990). To generate toluene vapor, the liquid material was heated, and the vapor diluted with nitrogen and mixed with the chamber ventilation air. An interim sacrifice was carried out at 15 months on control and 1200-ppm groups (10/sex/group) to conduct hematology and histopathology of the brain, liver, and kidney. Body weights were measured throughout the study. Gross necropsy and micropathology examinations were performed at the end of the study on all major organs including the nasal passage tissues (three sections), lungs, and mainstem bronchi. Mean body weights in both exposed groups were not different from controls for either sex. No exposure-related clinical signs were reported, and survival rate was similar for all groups. At the interim sacrifice, there was a mild-to-moderate degeneration in the olfactory and respiratory epithelium of the nasal cavity in 39/40 rats of the 600- and 1200-ppm groups compared with 7/20 controls. At the end of 2 years, there was a significant ( $p < 0.05$ ) increase in the incidence of erosion of the olfactory epithelium (males: 0/50, 3/50, and 8/49; females: 2/49, 11/50, and 10/50; at 0, 600, and 1200 ppm, respectively) and of degeneration of the respiratory epithelium (males: 15/50, 37/50, and 31/49; females: 29/49, 45/50, and 39/50; at 0, 600, and 1200 ppm, respectively) in the exposed animals. The females exposed to 600 and 1200 ppm also exhibited a significant increase in inflammation of the nasal mucosa (27/49, 42/50, and 41/50 at 0, 600, and 1200 ppm, respectively) and respiratory metaplasia of the olfactory epithelium (0/49, 2/50, and 6/50 at 0, 600, and 1200 ppm, respectively). A LOAEL of 600 ppm toluene was determined for the concentration-dependent increase in erosion of the olfactory epithelium in male rats and the degeneration of the respiratory epithelium in both sexes. No NOAEL could be derived from this study.

o INHALATION RFD UNCERTAINTY :

UF -- An uncertainty factor of 10 is used to account for intraspecies variability and another factor of 10 for the use of a LOAEL. An additional factor of 3 is applied for data base deficiencies, including the lack of data and well-characterized laboratory animal exposures evaluating neurotoxicity and respiratory irritation.

o INHALATION RFD MODIFYING :

MF -- None  
FACTOR

o INHALATION RFD COMMENTS :

Toluene-induced neurotoxicity has been documented in humans over a broad spectrum of severity that correlates well with concentration. Numerous case studies on chronic toluene abusers [repeatedly exposed to greater than 30,000 ppm (113,000 mg/cu.m)] have demonstrated functional deficits of the CNS accompanied by abnormal morphology of cerebellar and cortical areas of the brain. Under acute exposure conditions [short exposures to greater than 10,000 ppm (37,690 mg/cu.m)], toluene produces CNS narcosis [American Conference of Governmental Industrial Hygienists (ACGIH), 1991]. Lower concentrations, i.e., 800-400 ppm (3015-1508 mg/cu.m), have been associated with worker complaints of CNS-related effects (ACGIH, 1991). Clinical studies using controlled exposure to toluene have demonstrated concentration-related occurrence of complaints such as drowsiness, ataxia, visual impairment, and

headache. A number of occupational studies indicate that these same effects are present in exposed worker populations at concentrations lower than 400 ppm (1508 mg/cu.m) although deficiencies in most of these studies preclude confirming this finding unequivocally. Descriptions of a number of these studies follow. The preponderance of the literature showing CNS effects and the well-known proclivity for solvents to affect CNS processes in humans leave little doubt that the brain is a principal target organ for toluene toxicity in humans.

In cases of inhalation abuse of toluene, Rosenberg et al. (1988) demonstrated diffuse cerebral, cerebellar, and brainstem atrophy in 3 of 11 toluene abusers who also had neurological abnormalities. Filley et al. (1990) were able to correlate neuropsychological impairment with the degree of white matter abnormality ( $p < 0.01$ ). Cerebellar and cortical functions were classified as impaired in 15/24 individuals who had abused toluene daily (425 +/- 366 mg/day) for extended periods (6.3 +/- 3.9 years) (Fornazzari et al., 1983). In a limited case study, Metrick and Brenner (1982) demonstrated brainstem atrophy through computerized tomographic scans and abnormal brainstem auditory-evoked potentials in 2/2 chronic toluene abusers (12-16 years of admitted, continuous abuse). These studies confirm the occurrence of severe CNS damage in response to highly abusive concentrations of toluene.

Several studies that have investigated the occurrence of neurotoxicity at lesser concentrations, such as occupational situations, have not demonstrated significant neurological or other effects. Hanninen et al. (1987) performed a battery of 11 psychological tests on 43 printing workers who had been occupationally exposed to approximately 117 ppm (441 mg/cu.m) toluene for an average of 22 years and found only mildly adverse effects in 2/11 tests. The control and exposed cohorts in this study were, however, mismatched in several areas, most notably alcohol use. Iregren (1982) examined the psychological performance of 38 printers who had been occupationally exposed to 50-150 ppm (188-565 mg/cu.m) toluene for an average of 16.3 years (range 3-32 years). No effects were seen, although the cohorts in this study were apparently matched only by age. In a cohort study, Cherry et al. (1985) attempted to better match the control and exposed cohorts and considered alcohol use. Although no differences between the cohorts were statistically significant, the exposed workers performed worse than the nonexposed workers on 10/13 psychological tests. The 52 workers in this study were not, however, rigorously matched, and the concentrations listed in the study ranged up to greater than 500 ppm (1884 mg/cu.m). The cohorts in the study of Foo et al. (1990) were well matched for a number of confounders, including alcohol use, and statistically significant psychological effects were seen.

In the occupational study conducted by Yin et al. (1987), 94 solvent workers (38 men and 56 women; average employment duration, 6.8 years) and 138 controls (48 men and 90 women) were examined for exposure using diffusion dosimeters, subjective symptoms by questionnaire, hematology, and urinalysis. Exposure concentration (7-hour mean TWA) in the workers was estimated at 42.8 ppm (161 mg/cu.m) toluene with a maximum measurement of 123 ppm (464 mg/cu.m). Workers were co-exposed to 1.3 ppm benzene. No exposure-related effects were noted in any of the biochemical tests examined. In considering the prevalence of subjective symptoms (sore throat, headaches, and dizziness) workers were subgrouped into low (6-39 ppm,  $n = 28$ ) and high (40-123 ppm,  $n = 29$ ) categories. Although the prevalence of subjective symptoms was significantly higher in the exposed workers compared with the control cohort ( $p < 0.01$ ), a concentration-response relationship was not discernable among the groups. No other treatment-related effects were reported. The study was limited because the exposed and unexposed groups were not matched to control for confounding effects (e.g., age, smoking, alcohol consumption, exposure duration). Based on these results, exposure to an average of approximately 42.8 ppm toluene produced no biochemical abnormalities, although neither respiratory irritation nor psychological performance was directly evaluated in these workers.

In the occupational study by Lee et al. (1988), prevalence of subjective symptoms was categorized with respect to exposure levels. The study population (193 women and 65 controls) completed a questionnaire. The exposures were reported as 8-hour TWAs, and workers were grouped in exposure categories of nonexposed, 1-50 ppm, 51-100 ppm, 101-150 ppm, and more than 151 ppm (duration of exposures was not reported). A concentration-dependent

increase in prevalence was reported for 25/67 symptoms with increases in complaints over controls occurring at around 100 ppm (348 mg/cu.m). Similar to the Yin et al. study (1987) reported above, symptomatology included headaches, sore throats, and dizziness. Although an effect level in humans of around 100 ppm is indicated by this study, no objective measures of toxicity were examined.

A number of acute human studies have focused on toluene effects. In general, these studies corroborate subjective CNS effects such as headaches and dizziness reported in other longer-term occupational studies (Yin et al., 1987; Lee et al., 1988) and also document irritation effects. The study of Echeverria et al. (1989) correlates the occurrence of these subjective effects with substantial neurological symptoms.

Forty-two college students (21 female and 21 male) were exposed to 0, 74 ppm (279 mg/cu.m), or 151 ppm (569 mg/cu.m) toluene for 7 hours over 3 days (Echeverria et al., 1989). This exposure sequence was repeated for a total of 42 exposures over a 3-month period. The odor of toluene was masked. A battery of performance tests was administered to each participant prior to starting the exposures and again at 4 and 7 hours during the exposure; the initial test served as a control for those tests performed during the exposure. A 5-10% decrement in performance was considered significant if consistent with a linear trend. Test results for visual perception differed from control values for both exposure levels. Results of a manual dexterity test differed from control values at the higher but not the lower exposure level. Psychomotor test results were unaffected by toluene exposure. Subjective symptomatology increased with exposure with increasing numbers of complaints of eye irritation, headache, and somnolence. A NOAEL of 74 ppm (279 mg/cu.m) is indicated for these results. The duration-adjusted value is 122 mg/cu.m for these acute effects.

Andersen et al. (1983) exposed 16 subjects (average age of 24 years) to 0, 10, 40, or 100 ppm (0, 38, 151, or 377 mg/cu.m) toluene for 6 hours on each of 4 consecutive days. Individuals were tested for nasal mucous flow, lung function, subjective response, and psychometric performance. At 100 ppm, irritation was experienced in the eyes and nose, but no effect on nasal mucous flow or lung function was observed. The subjects frequently reported headaches, dizziness, and a feeling of intoxication. These effects were not reported by the 10- or 40-ppm exposure groups. No effects were seen in performance tests. This study indicates an effect level of 100 ppm, and a NOAEL of 40 ppm (151 mg/cu.m).

The acute study by Baellum et al. (1990) evaluated 32 males and 39 females exposed to 0 or 100 ppm (0 or 377 mg/cu.m), or to varying exposures of 50-300 ppm (188-1131 mg/cu.m) (TWA = 102 ppm), for 7 hours. Volunteers exercised on an ergometer cycle for 3 periods of 15 minutes each during the exposure. No significant differences were found in the performances between the exposed and control groups in a battery of tests for performance, visual attention, and reaction times. Exposed subjects reported an increase over nonexposed subjects ( $p < 0.1$ ) in nose and lower respiratory irritation, feelings of intoxication, dizziness, increased coughing, and headaches. Differences were not noted between the group exposed to a constant level (100 ppm) and the group exposed to the same TWA, but with peaks of up to 300 ppm.

Baellum et al. (1985) investigated the effects of a 6.5-hour toluene exposure to 43 printers with a long-term occupational exposure to a mixture of solvents including toluene and 43 controls with no history of exposure to solvents or other chemicals. The duration of employment for the workers ranged from 9-25 years. Each individual was exposed only once to either 0 or 100 ppm (0 or 377 mg/cu.m) toluene during a 6.5-hour exposure period, preceded by a 1-hour acclimatization period. These subjects were then subgrouped into printers exposed to toluene ( $n = 20$ ), printers exposed to air ( $n = 23$ ), controls exposed to toluene ( $n = 21$ ), and controls exposed to air ( $n = 22$ ). All subjects carried out a battery of tests for psychometric performance, visual perception, and vigilance evaluation. Both printers and controls complained of nasal and eye irritation, unacceptable air quality, and unacceptable odor level during the toluene exposure. Signs of neurotoxicity, including moderate fatigue, sleepiness, headaches, and a feeling of intoxication, were likewise similarly reported for both groups. A significant

decrease in performance was found for the pegboard visual motor function test in the exposed printers, but not in the controls exposed to 100 ppm toluene. A decrease in psychometric performance, primarily in visual perception and accuracy, was observed in toluene-exposed individuals. Acute exposure to toluene resulted in a lower performance in 4/10 tests conducted, 3 of these 4 evaluated visual perception. The most profound difference between subjects exposed to 100 ppm toluene and those exposed to clean air was observed in the color discrimination test; this difference was seen in both exposed vs. nonexposed printers and exposed vs. nonexposed controls. This study indicates that little tolerance develops to the irritative and central effects in humans exposed to toluene and that 100 ppm (377 mg/cu.m) is the effect level for these symptoms.

Von Ottingen et al. (1942) exposed 3 humans to 100 or 200 ppm (377 or 754 mg/cu.m) toluene vapors for 8 hours. At 200 ppm, the subjects experienced muscular weakness, confusion, impaired coordination, and dilated pupils, with after-effects including fatigue, general confusion, and moderate insomnia. In 1 subject exposed to 100 ppm toluene, moderate fatigue, sleepiness, and headaches were reported.

Hepatotoxicity has also been examined as a toxicologic endpoint of toluene exposure in humans. Fornazzari et al. (1983) described moderate elevation of serum AP levels in 13/24 (and SGOT in 7/24) toluene abusers upon admission to a clinic. These elevated levels were normal after 2 weeks of solvent abstinence, although the accompanying CNS effects were only minimally improved. In a cross-sectional study of 181 printing workers in which toluene exposures were less than 200 mg/cu.m, no adverse effects were apparent as judged from serum liver enzymes (Boewer et al., 1988). In another cross-sectional occupational study conducted by Guzelian et al. (1988) that involved 289 printing factory employees, 8 workers were found who had an increase described as "marked" in the ratio of ALT/AST enzyme serum activity. Biopsies revealed mild pericentral fatty livers in each of the eight cases. Based on environmental data (probably area monitors) the levels of toluene to which these workers were exposed was less than 200 mg/cu.m., 2-8 hours/day.

Fischer 344 rats (120/sex/group) inhaled 0, 30, 100, or 300 ppm (0, 113, 377, or 1130 mg/cu.m, respectively) toluene (99.9% purity), 6 hours/day, 5 days/week (duration-adjusted to 0, 20, 67, or 202 mg/cu.m, respectively) for 106 weeks (CIIT, 1980; Gibson and Hardisty, 1983). Vapor, generated by bubbling clean air through toluene, was passed through the air supply duct and mixed with air by turbulent flow to produce the desired concentration. Hematology, blood chemistry, and urinalysis were conducted in all groups at 6 (5/sex), 17 (5/sex), 18 (10-20/sex), and 24 months (10/sex). Histopathology was evaluated only in the control and 300-ppm groups at 6 (5/sex), 12 (5/sex), and 18 months (20/sex). At 24 months, histopathological examinations were conducted in organs of all surviving animals, including the respiratory system and sections through the nasal turbinates (number not indicated). No treatment-related non-neoplastic effects were observed in the exposed animals. Although the male rats exposed to 300 ppm had a significant increase in body weight compared to controls, no concentration-response was evident. At the end of the exposure period, the female rats exposed to 100 or 300 ppm exhibited a slight but significant reduction in hematocrit; an increase in the mean corpuscular hemoglobin concentration was also noted but only in the females exposed to 300 ppm. The highest concentration examined in this study, 300 ppm, is designated as a NOAEL for toxicity remote from the respiratory tract in rats. CIIT (1980) reported that the technical and raw data were not audited by their quality assurance group during the study period, although CIIT did conduct a quality assessment procedure to review the data. The available pathology reports containing these data indicate that at least the lower respiratory tract was examined. Communication with the testing sponsor has provided information indicating that only one section was examined from the nasal cavity of these test animals. It is not clear whether this single section would have been sufficient to elucidate the areas of lesions noted in the NTP (1990) study. Consequently, the designation of the 300-ppm exposure level as a NOAEL for respiratory lesions (see NTP, 1990) is problematic.

Fischer 344/N rats (10/sex/group) were exposed to toluene vapors at 0, 100, 625, 1250, 2500, and 3000 ppm (0, 377, 2355, 4711, 9422, and 11,307 mg/cu.m, respectively) 6.5 hours/day, 5 days/week (duration-adjusted to 0, 73,

455, 911, 1823, and 2187 mg/cu.m, respectively) for 15 weeks (NTP, 1990). Organ weights were measured and histological examinations were performed only on controls, 2500- and 3000-ppm groups, and animals that died before the end of the study. Eight of 10 males exposed to 3000 ppm died, all during the 2nd exposure week. No females died at any exposure level. Compared to the controls, final body weights were 15 and 25% lower in the males and 15 and 14% lower in the females of the 2500- and 3000-ppm groups, respectively. There was a concentration-related increase in the relative liver weight, significant at 1250, 2500, and 3000 ppm in males and at 2500 and 3000 ppm in females. The relative weights of the heart, lung, kidney, and right testis were also significantly elevated in the 2500- and 3000-ppm animals compared to those of the controls, although no histopathology was observed in any exposure group. Toxic effects noted in a concurrently conducted gavage study (urinary bladder hemorrhages in the two highest exposure groups) were not noted in this subchronic inhalation study. A LOAEL of 2500 ppm [LOAEL(HEC) = 1823 mg/cu.m] was determined for the decrease in body weight gain in both males and females, and the NOAEL for this effect was 1250 ppm [NOAEL(HEC) = 911 mg/cu.m].

Toluene has been suspected to cause congenital defects in infants born to mothers who were exposed to or who abused toluene during pregnancy. In a case report study, Hersh et al. (1985) describes clinical and morphometric characteristics common to 3 children whose mothers had abused toluene (but apparently not alcohol or any other substance) for a period of 4-5 years including during their pregnancies with the affected children. Clinical findings common to these three children included microcephaly, CNS dysfunction, attention deficits, and developmental delay/mental deficiency. Phenotypic similarities included a small midface, deep-set eyes, micrognathia (smallness of the jaws), and blunting of the fingertips. A retrospective cohort study was conducted by McDonald et al. (1987) who examined the history of exposure to chemicals of 301 women who had recently given birth to an infant with an important congenital defect. An identical number of women (referents) who had given birth to normal children were matched with respect to age, employment (hours/week), date of delivery, and educational level. In initial matched-pair analysis, chemical exposure was higher in the cases than in the referents (63 cases:47 referents) due to excess cardiac and miscellaneous defects. In further analysis by chemical categories, only exposure to aromatic solvents showed a clear excess of defects, mostly in the urinary tract. Details of these cases (n = 19) showed that toluene was identified as the solvent in 11 of these cases.

Mudak and Ungvary (1978) exposed three groups of pregnant CFY rats to toluene during different periods of gestation and for different durations of exposure. Two of the groups had their own control group exposed to air only and matched for period and daily duration. The first of these (n = 19) was exposed to 1500 mg/cu.m for 24 hours/day during gestational days 9 to 14. Two dams died during these exposures. No details on the deaths are given but no other maternal toxicity was observed. Fetotoxicity was also in evidence as sternebral alterations (6% vs. 1% in controls), extra ribs (22% vs. 0% in controls), and the presence of fetuses with missing tails (2/213, none observed in 315 controls) were recorded. Under these exposure conditions, 1500 mg/cu.m is a LOAEL for fetotoxicity and a frank effect level (FEL) for maternal toxicity. The second group (n = 14) received this same concentration continuously but on days 1-8 of gestation. Five dams died under these exposure conditions although toxicity parameters of the surviving dams were identical with the controls from the first group (gestational days 9-14). Slight hydrocephaly was noted in 4 fetuses (all from the same litter), and 17% growth retardation was noted vs. 7% in the controls. Thus these exposure conditions are a FEL for maternal toxicity and a LOAEL for fetotoxicity. A third group was exposed to 1000 mg/cu.m for 8 hours/day from the 1st to the 21st day of gestation. No maternal deaths or toxicity occurred. Minor skeletal retardation was present in the exposed fetuses at a higher incidence rate (25%) than in concurrent controls (0%). These results indicate that 1000 mg/cu.m is a LOAEL for developmental effects under these exposure conditions. This concentration is also a NOAEL for maternal effects. These workers also exposed groups of pregnant CFLP mice (n = 11-15) to either air or 1500 or 500 mg/cu.m toluene continuously during days 6-13 of pregnancy. All mice exposed to the high concentration died within 24 hours of the beginning of exposure. No dams died in the lower exposure group. In this group, the average fetal weight decreased to 0.96 g from the average control weight of 1.07 g, and the

percentage of weight-retarded fetuses (less than 0.9 g) increased to 27.6% from 6.5% in the controls. No difference in incidence of skeletal malformations or anomalies was noted between these and control fetuses. For mice, 1500 mg/cu.m is an FEL and 500 mg/cu.m is a mild LOAEL. Since duration adjustment is not performed for developmental effects, this concentration is also the LOAEL(NEC).

B6C3F1 mice (60/sex/group) were exposed to 0, 120, 600, or 1200 ppm (0, 452, 2261, or 4523 mg/cu.m, respectively) toluene 6.5 hours/day, 5 days/week (duration-adjusted to 0, 87, 47, and 875 mg/cu.m, respectively) for 2 years (NTP, 1990). Mean body weights were not significantly different among groups and no treatment-related clinical signs were observed. Deaths (moribund and natural) occurred in all exposure groups but were not related to exposure and were not greater than the control rates. An excess incidence of non-neoplastic inflammatory lesions of the urinary and genital system was observed in all the groups of male mice. At the 15-month interim sacrifice, minimal hyperplasia in the bronchial epithelium was observed in 4/10 females exposed to 1200 ppm. At the end of the study, there was a concentration-dependent increase in the incidence of splenic pigmentation in the exposed males (9/60, 11/60, and 18/59 at 120, 600, and 1200 ppm, respectively) compared to controls (4/60). In the females, the incidence was 37/50, 33/50, 34/49, and 28/47 at 0, 120, 600, and 1200 ppm, respectively. The occurrence of endometrial hyperplasia was present in 14% of the animals exposed to the highest concentration but only in 4% in the low-exposure groups and controls. No differences were noted between the exposed and control mice of either sex in the incidence of degeneration of either the olfactory or respiratory epithelium. No other non-neoplastic lesions were observed in exposed mice. As no adverse effects were noted in this study, the highest concentration, 1200 ppm was designated as a NOAEL in mice for this chronic study [NOAEL(NEC) = 875 mg/cu.m].

Sprague-Dawley rats (15/sex/group) were exposed to cumulative mean exposures of 0, 100, or 1481 ppm (0, 377, or 5653 mg/cu.m) toluene vapors, 6 hours/day, 5 days/week (duration-adjusted to 0, 67, and 1009 mg/cu.m, respectively) for 26 weeks (API, 1981). On weeks 9, 18, and 27, neurohistopathological examinations were conducted in 3-5 rats/sex/group. Hematology, clinical chemistry, and urinalysis parameters were evaluated after 13 and 26 weeks of exposure. Body weights were measured weekly. No significant treatment-related effects were reported. Therefore, a NOAEL of 1481 ppm [NOAEL(NEC) = 1009 mg/cu.m] toluene was determined for systemic effects in rats. The study was limited because there were no other neurohistopathological examinations or organ weight measurements conducted on the animals.

Inhalation exposure to toluene has been shown to result in irreversible high-frequency hearing loss in rats. Pryor et al. (1984) exposed young male Fischer 344 rats to a variety of exposure concentrations and durations. Hearing loss was evaluated by a behavioral technique (avoidance response elicited to an auditory signal) or brainstem auditory-evoked responses (elicited by tone pips of differing loudness and frequency and detected by subdural scalp electrodes). Hearing loss, as measured by both techniques, was observed after as few as 2 weeks exposure to 1000 ppm toluene for 14 hours/day. Lower concentrations of 700 ppm for 14 hours/day were without effect after 16 weeks of exposure. Intermittent exposure to 3000 ppm for 30 minutes/hour for 8 hours/day caused hearing loss within 2 weeks, whereas a similar exposure schedule for only 4 hours/day was without effect after 9 weeks. These data define a NOAEL for hearing loss in rats of 700 ppm [NOAEL(NEC) = 2638 mg/cu.m]. The duration-adjusted NEC (assumed 5 days/week) would be 14/24 hours x 5/7 days = 1100 mg/cu.m. Although these results clearly document hearing loss in young adult rats, their direct significance to humans remains unclear. Among chronic toluene abusers there is only a single report of adverse effects on hearing; Metrick and Brenner (1982) claimed that the abnormal auditory-evoked potentials recorded in two chronic toluene abusers was evidence of brainstem abnormalities.

Pregnant Wistar rats and hamsters (group size not indicated) inhaled 0 or 800 mg/cu.m toluene vapors 6 hours/day on gestational days 14-20 (rats) or gestational days 6 to 11 (hamsters) (DeSilva et al., 1990). In the exposed rats, there was a significant ( $p < 0.05$ ) increase in the number of litters with one or more low birth weight pups (less than 4.9 g), from 10% in the controls

to 54% in the exposed dams. A decrease ( $p < 0.05$ ) in the number of live pups at birth was also noted in the litters of exposed dams. No evaluation of malformations or anomalies was performed. The neurobehavioral development of the offspring of the exposed rats was assessed using tests of spontaneous alternation, rim escape, and avoidance responses. The only effect noted in the rats, a shortened first trial latency in choosing one side of a maze, was minimal and its significance unclear. No comparable reproductive deficits occurred in the exposed hamsters. The only effect noted in the neurobehavioral tests of the hamster offspring was an equivocal effect in rota-rod performance. No neurobehavioral effect levels were designated from this study, although it appears that the rat developmental processes are more sensitive than those of the hamster, exhibiting adverse effects at 800 mg/cu.m.

Ungvary and Tatrai (1985) exposed New Zealand rabbits (8-10/group) to 0, 500, or 1000 mg/cu.m toluene, 24 hours/day, on gestational days 7-20, and CFLP mice (15 females/group) to 0, 500, 1000, or 1500 mg/cu.m toluene, also continuously, on gestational days 6-15. The control groups consisted of 115 mice and 60 rabbits. All the female mice exposed to 1500 mg/cu.m died. In the mice exposed to 1000 mg/cu.m, there was an increase in fetuses with retarded weight (29%, level of retardation not indicated) and in fetuses with skeletal retardation (12%) compared to 7% and 5%, respectively, in the controls, which did not differ from the animals exposed to 500 mg/cu.m. Of the 8 pregnant rabbits exposed to 1000 mg/cu.m, 2 died, 4 had spontaneous abortions, and the remaining 2 had total litter resorption. No deaths occurred in the 10 rabbits exposed to 500 mg/cu.m but 1/10 rabbits had a spontaneous abortion (as compared to 0/60 reported for the controls). A NOAEL(HEC) of 500 mg/cu.m toluene was determined for reproductive effects in mice. For rabbits, the 500 mg/cu.m concentration is designated as a LOAEL. These results indicate that pregnant mice may be a sensitive population to the effects of toluene.

Pregnant Charles River CD-1 mice (15-16 females/group) inhaled filtered air or 200 or 400 ppm (754 and 1508 mg/cu.m) toluene 7 hours/day on gestational days 7-16 (Courtney et al., 1986). The relative liver weight in the exposed dams was reported to be significantly lower in the two exposed groups compared to the controls, although no data were presented. A statistically significant increase in lactate dehydrogenase activity in the brain of the dams exposed to 400 ppm was also reported. The exposed pregnant mice did not exhibit any significant differences in the number of implantation sites, number of live fetuses, fetal deaths, or fetal body weight compared to the control values. A statistically significant increase over controls in the incidence (both per litter and per fetus) of enlarged renal pelvis was noted in dams exposed to 200 ppm but not 400 ppm. A statistically significant alteration from controls in the rib profile (percentage of fetuses with 1 or 2 additional/fewer ribs) was reported for fetuses from dams exposed to 400 ppm but not 200 ppm. The toxicological significance of this finding is not clear. As no clearly significant toxicological effects were observed, the highest concentration used, 400 ppm (NOAEL(HEC) = 1508 mg/cu.m) is designated as a NOAEL for reproductive and developmental effects in mice.

A 2-generation inhalation reproductive study was conducted in CD rats (10-40 males, 20-80 females/group) (API, 1985). Animals were exposed by whole-body inhalation to toluene at 0, 100, 500, or 2000 ppm (0, 377, 1885, or 7538 mg/cu.m, respectively) 6 hours/day, 7 days/week for 80 days and a 15-day mating period. The mated females were then exposed to the same concentrations during days 1-20 of gestation and days 5-20 of lactation. After weaning, the pups in this generation (F1) were exposed 80 times and then randomly mated with members of the same exposure group (2 females/1 male) to produce the second generation (F2). Mean male body weights were slightly reduced (maximum of 10%) in the first 2 weeks of the exposure in the animals exposed to 500 and 2000 ppm, although the size of the reduction was not related to exposure. No differences were observed in male or female fertility indices, length of gestation, mean numbers of viable and nonviable pups at birth, or pup survival indices during lactation. No abnormal histopathology was noted in organs examined. A significant decrease ( $p < 0.05$ ) in weight relative to controls was observed in the first generation offspring. The decrease was maintained throughout the lactation period in the pups from dams exposed to the highest exposure and in those from the ancillary group in which females exposed to the 2000 ppm concentration were mated with males having no exposure. No data were



available in the report about the F2 generation. Based on the effects on the pups of the first generation (F1), a LOAEL of 2000 ppm [LOAEL(HEC) = 7538 mg/cu.m] is designated, the NOAEL being 500 ppm [NOAEL(HEC) = 1885 mg/cu.m].

o INHALATION RFD CONFIDENCE : Study -- Medium Data Base -- Medium RfC -- Medium The study of Foo et al. (1990) indicates adverse neurological effects of toluene in a small worker population. These effects are consistent with more severe CNS effects occurring at abusive concentrations of toluene and could not have been confounded by alcohol as the control and exposed populations did not use alcohol. However, the paucity of exposure information and identification of only a LOAEL is not sufficient to warrant a higher confidence than medium for this study. Other studies indicate that irritation may occur at around the same concentration, 100 ppm (Baelum et al., 1985; Echeverria et al., 1989). In regard to this effect, the NTP (1990) rat chronic inhalation study was well conducted, established the rat as the most sensitive species, examined an adequate number of animals, and performed histopathology on all major organs, including the brain and the respiratory tract. The sensitive endpoint was the concentration-dependent degeneration of the nasal epithelium characterized by the erosion of the olfactory epithelium and degeneration of the respiratory epithelium in male rats. The NTP study is also given medium confidence, however, as it did not establish a NOAEL. Although this data base has a complement of chronic laboratory animal studies, long-term data in humans are not available for either the neurotoxicity or irritation endpoints. The reproductive/developmental studies in three species were not comprehensive in endpoint evaluation but do identify the rabbit as the most sensitive species. The data base is thus given a medium confidence rating. A medium confidence rating for the RfC follows.

o INHALATION RFD SOURCE :

Source Document -- This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation -- U.S. EPA, 1984, 1985  
DOCUMENT

o REVIEW DATES : 04/21/88, 05/26/88, 02/16/89, 03/21/89,  
05/18/89, 08/15/91, 12/11/91  
o VERIFICATION DATE : 05/18/89, 12/11/91  
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CAREV-

o CLASSIFICATION : D; not classified  
o BASIS FOR CLASSIFICATION : No human data and inadequate animal data.  
Toluene did not produce positive results in the majority of genotoxic assays.

o HUMAN CARCINOGENICITY DATA :

None.

o ANIMAL CARCINOGENICITY DATA :

A chronic (106-week) bioassay of toluene in F344 rats of both sexes reported no carcinogenic responses (CIIT, 1980). A total of 960 rats were exposed by inhalation for 6 hours/day, 5 days/week to toluene at 0, 30, 100, or 300 ppm. Groups of 20/sex/dose were sacrificed at 18 months. Gross and microscopic examination of tissues and organs identified no increase in neoplastic tissue or tumor masses among treated rats when compared with controls. The study is considered inadequate because the highest dose administered was well below the MTD for toluene and because of the high incidence of lesions and pathological changes in the control animals.

Several studies have examined the carcinogenicity of toluene following repeated dermal applications. Toluene (dose not reported) applied to shaved interscapular skin of 54 male mice (strains A/He, C3HeB, SWR) throughout their lifetime (3 times weekly) produced no carcinogenic response (Poel, 1963). One drop of toluene (about 6 mL) applied to the dorsal skin of 20 random-bred albino mice twice weekly for 50 weeks caused no skin papillomas or carcinomas after a 1-year latency period was allowed (Coombs et al., 1973). No increase in the incidence of skin or systemic tumors was demonstrated in male or female mice of three strains (CF, C3H, or CBA/N) when toluene was applied to the back of 25 mice of each sex of each strain at 0.05-0.1 mL/mouse, twice weekly for 56 weeks (Doak et al., 1976). One skin papilloma and a single skin carcinoma were reported among a group of 30 mice treated dermally with one drop of 0.2% (w/v) solution toluene twice weekly, administered from droppers delivering 16-20  $\mu$ L per drop for 72 weeks (Lijinsky and Garcia, 1972). It is not reported whether evaporation of toluene from the skin was prevented during these studies.

o SUPPORTING DATA :

Toluene was found to be nonmutagenic in reverse mutation assays with *S. typhimurium* (Mortelmans and Riccio, 1980; Nestmann et al., 1980; Bos et al., 1981; Litton Bionetics, Inc., 1981; Snow et al., 1981) and *E. coli* (Mortelmans and Riccio, 1980), with and without metabolic activation. Toluene did not induce mitotic gene conversion (Litton Bionetics, Inc., 1981; Mortelmans and Riccio, 1980) or mitotic crossing over (Mortelmans and Riccio, 1980) in *S. cerevisiae*. Although Litton Bionetics, Inc. (1981) reported that toluene did not cause increased chromosomal aberrations in bone marrow cells, several Russian studies (Dobrokhotov, 1972; Lyspkalo, 1973) report toluene as effective in causing chromosomal damage in bone marrow cells of rats. There is no evidence of chromosomal aberrations in blood lymphocytes of workers exposed to toluene only (Maki-Paakkanen et al., 1980; Forni et al., 1971), although a slight increase was noted in workers exposed to toluene and benzene (Forni et al., 1971; Funes-Craviota et al., 1977). This finding is supported by studies of cultured human lymphocytes exposed to toluene in vitro; no elevation of chromosomal aberrations or sister chromatid exchanges was observed (Gerner-Smidt and Friedrich, 1978).

CARDR-

o CARCINOGENICITY SOURCE :

U.S. EPA. 1987. Drinking Water Criteria Document for Toluene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. ECAO-CIN-408.

The values in the 1987 Drinking Water Criteria Document for Toluene have received peer and administrative review.

DOCUMENT

o REVIEW DATES : 09/15/87  
o VERIFICATION DATE : 09/15/87  
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MAONE-

One-day HA -- 2E+1 mg/L

NOAEL -- 21.5 mg/kg/day

UF -- 10 (allows for intrahuman variability with the use of a NOAEL from a human study)

Assumptions -- 1 L/day water consumption for a 10-kg child

Principal Study -- Gamberale and Multengren, 1972

This study reported that a 20-minute exposure to 100 ppm toluene was a no-effect level when determined by perceptual speed and reaction time tests in human volunteers. At 200 ppm, toluene was noted as clearly causing toxic effects such as incoordination, exhilaration, and prolonged reaction time. These and other data support the selection of 100 ppm (377 mg/cu.m) toluene as the NOAEL in humans exposed for up to 8 hours. Based on the conditions of exposure and an assumed absorption rate of 60%, this level is equivalent to 21.5 mg/kg/day.

HATEN-

No information was found in the available literature that was suitable for determination of a Ten-day HA value. It is, therefore, recommended that the DWEL, adjusted for a 10-kg child (3 mg/L) be used as the Ten-day HA value.

HALTC-

No information was found in the available literature that was suitable for determination of a Longer-term HA value. It is, therefore, recommended that the DWEL, adjusted for a 10-kg child (3 mg/L) be used as the Longer-term HA value for a child.

HALTA-

No information was found in the available literature that was suitable for determination of a Longer-term HA value. It is, therefore, recommended that the DWEL, adjusted for a 70-kg adult (10 mg/L) be used as the Longer-term HA value for an adult.

HALIF-

Drinking Water Equivalent Level (DWEL) -- 7E-0 mg/L

Assumptions -- 2 L/day water consumption for a 70-kg adult

RfD Verification Date -- 06/20/90

Lifetime HA -- 1E-0 mg/L

Assumptions -- 20% exposure by drinking water

Principal Study -- NTP, 1989 (This study was used in the derivation of the chronic oral RfD; see RDO)

-----  
OLEP -

Taste threshold in water is reported as 0.04 and 1 mg/L. Odor threshold in water is reported as 0.04 and 1 mg/L.  
-----

ALAB -

Analysis of toluene is by a purge-and-trap gas chromatographic procedure used for the determination of volatile aromatic and unsaturated organic compounds in water.  
-----

TREAT-

Treatment options for removing toluene from drinking water sources include air stripping and adsorption onto granular activated carbon.  
-----

HADR -

o HEALTH ADVISORY SOURCE :

U.S. EPA. 1990. Final Draft of the Drinking Water Criteria Document for Toluene. Office of Drinking Water, Washington, DC.  
DOCUMENT  
-----

o HEALTH ADVISORY REVIEW :

EPA review of HAs in 1986.

Public review of HAs in 1987.

Science Advisory Board review to be determined.  
-----

o EPA DRINKING WATER CONTACT :

Krishan Khanna / OST -- (202)260-9568

Edward V. Ohanian / OST -- (202)260-7571  
-----

WQCHU-

Water and Fish Consumption: 1.43E+4 ug/L

Fish Consumption Only: 4.24E+5 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The WQC of 1.43E+4 ug/L is based on consumption of contaminated aquatic organisms and water. A WQC of 4.24E+5 ug/L has also been established based on consumption of contaminated aquatic organisms alone.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OURS  
(202)260-1315 / FTS 260-1315  
-----

WQCAQ-

Freshwater:

Acute LEC -- 1.75E+4 ug/L

Chronic LEC -- none

Marine:

Acute LEC -- 6.3E+3 ug/L  
Chronic LEC -- 5.0E+3 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LEC's are given when the minimum data required to derive water quality criteria are not available.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWS  
(202)260-1315 / FTS 260-1315

.....  
.....  
.....  
MCLG -

Value (status) -- 1 mg/L (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion -- EPA has set a MCLG for toluene based on its potential adverse effects reported in a 13-week oral study in rats. The MCLG is based upon a DWEL of 7 mg/L and an assumed drinking water contribution of 20 percent.

Reference -- 54 FR 22062 (05/22/89)

EPA Contact -- Health and Ecological Criteria Division / OST /  
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

.....  
.....  
MCL -

Value -- 1 mg/L (Final, 1991)

Considers technological or economic feasibility? -- YES

Monitoring requirements -- All systems initially monitored for four consecutive quarters; repeat monitoring dependent upon detection, vulnerability status and system size.

Analytical methodology -- Gas chromatography (EPA 502.2, 503.1);  
gas chromatography/mass spectrometry (EPA 524.1, 524.2); PQL= 0.005 mg/L.

Best available technology -- Granular activated carbon; packed tower aeration

Reference -- 56 FR 3526 (01/30/91); 56 FR 30266 (07/01/91)

EPA Contact -- Drinking Water Standards Division / OGDW /  
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

\_\_\_IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

Value -- 0.04 mg/L (Proposed, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- SMCLs are non-enforceable and establish limits for contaminants which may affect the aesthetic qualities (e.g. taste and odor) of drinking water. It is recommended that systems monitor for these contaminants every three years. More frequent monitoring for contaminants such as pH, color, odor or others may be appropriate under certain circumstances. The SMCL for toluene

is based on odor detection. Promulgation deferred following public comment (56 FR 3526).

Reference -- 54 FR 22062 (05/22/89); 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGDW /  
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

#### IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

CERC -

Value (status) -- 1000 pounds (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ is based on aquatic toxicity, as established under Section 311(b)(4) of the Clean Water Act, ignitability, and chronic toxicity. Available data indicate that the aquatic 96-Hour Median Threshold Limit for Toluene is between 10 and 100 ppm. Its closed-cup flash point is less than 100F and its boiling point is >100F. RQ assignments based on chronic toxicity reflect two primary attributes of the hazardous substance, the minimum effective dose (MED) levels for chronic exposure (mg/day for a 70-kg person) and the type of effect (liver necrosis, teratogenicity, etc). A composite score is determined from an evaluation of these two attributes. Toluene was determined to have a composite score between 6 and 20, corresponding to a chronic toxicity RQ of 1000 pounds.

Reference -- 50 FR 13456 (04/04/85); 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

- OREF - CIIT (Chemical Industry Institute of Technology). 1980. A 24-month inhalation toxicology study in Fischer-344 rats exposed to atmospheric toluene. CIIT, Research Triangle Park, NC.
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- OREF - Newrot, P.S. and R.E. Staples. 1979. Embryo-fetal toxicity and teratogenicity of benzene and toluene in the mouse. Teratology. 19: 41A

- (abstr.)
- OREF - NTP (National Toxicology Program). 1989. Toxicology and carcinogenesis studies of toluene (CAS No. 108-88-3) in F344/N rats and B6C3F1 mice (inhalation studies). Technical Report Series No. 371. Research Triangle Park, NC.
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  - IREF - ACGIH (American Conference of Governmental Industrial Hygienists). 1991. Notice of intended changes - toluene, trimethylamine, and vinyl acetate. Appl. Occup. Environ. Hyg. 6(11): 966-977.
  - IREF - Andersen, I., G.R. Lundqvist, L. Molhave et al. 1983. Human response to controlled levels of toluene in six-hour exposures. Scand. J. Work Environ. Health. 9: 405-418.
  - IREF - API (American Petroleum Institute). 1981. 26-Week inhalation toxicity study of toluene in the rat. Conducted by Bio/dynamics Inc. and Institute of Neurotoxicity, Albert Einstein College of Medicine for API, Washington, DC.
  - IREF - API (American Petroleum Institute). 1985. Two-generation inhalation reproduction/fertility study on a petroleum-derived hydrocarbon. Doc. ID FYI- AX-0284-0294 IN. Microfiche No. 0294.
  - IREF - Baelum, J., I. Andersen, G.R. Lundqvist et al. 1985. Response of solvent-exposed printers and unexposed controls to six-hour toluene exposure. Scand. J. Work Environ. Health. 11: 271-280.
  - IREF - Baelum, J., G. Lundqvist, L. Molhave and N.T. Andersen. 1990. Human response to varying concentrations of toluene. Int. Arch. Occup. Environ. Health. 62(1): 65-71.
  - IREF - Boewer, C., G. Enderlein, U. Wollgast, S. Nawka, H. Palowski, and R. Bleiber. 1988. Epidemiological study on the hepatotoxicity of occupational toluene exposure. Int. Arch. Occup. Environ. Health. 60: 181-186.
  - IREF - Cherry, N., H. Hutchins, T. Pace and M.A. Waldron. 1985. Neurobehavioral effects of repeated occupational exposure to toluene and paint solvents. Br. J. Ind. Med. 42(5): 291-300.
  - IREF - CIIT (Chemical Industry Institute of Toxicology). 1980. A twenty-four month inhalation toxicology study in Fischer-344 rats exposed to atmospheric toluene. Conducted by Industrial Bio-Test Laboratories, Inc., Decatur, IL, and Experimental Pathology Laboratories, Inc., Raleigh, NC, for CIIT, Research Triangle Park, NC. October 15, 1980.
  - IREF - Courtney, K.D., J.E. Andrews, J. Springer et al. 1986. A perinatal study of toluene in CD-1 mice. Fund. Appl. Toxicol. 6: 145-154.
  - IREF - DaSilva, V.A., L.R. Malheiros and F.M.R. Bueno. 1990. Effects of toluene exposure during gestation on neurobehavioral development of rats and hamsters. Brazil J. Med. Biol. Res. 23: 533-537.
  - IREF - Echeverria, D., L. Fine, G. Langolf, A. Schork and C. Sempio. 1989. Acute neurobehavioral effects of toluene. Br. J. Ind. Med. 46(7): 483-495.
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  - IREF - Foo, S.C., J. Jeyaratnam and D. Koh. 1990. Chronic neurobehavioral effects of toluene. Br. J. Ind. Med. 47(7): 480-484.
  - IREF - Fornazzari, L., D.A. Wilkinson, B.M. Kapur and P.L. Carlen. 1983. Cerebellar, cortical and functional impairment in toluene abusers. Acta Neurol. Scand. 67: 319-329.
  - IREF - Gibson, J.E. and J.F. Hardisty. 1983. Chronic toxicity and oncogenicity bioassay of inhaled toluene in Fischer-344 rats. Fund. Appl. Toxicol. 3: 315-319.
  - IREF - Guzelian, P., S. Mills and M.J. Fallon. 1988. Liver structure and function in print workers exposed to toluene. J. Occup. Med. 30(10): 791-796.
  - IREF - Hanninen, M., M. Antti-Poika and P. Savolainen. 1987. Psychological performance, toluene exposure and alcohol consumption in rotogravure printers. Int. Arch. Occup. Environ. Health. 59(5): 475-483.
  - IREF - Hersh, J.H., P.E. Podruch, G. Rogers and B. Weisskopf. 1985. Toluene embryopathy. J. Pediatr. 106: 922-927.
  - IREF - Hudak, A. and G. Ungvary. 1978. Embryotoxic effects of benzene and its methyl derivatives: Toluene, xylene. Toxicology. 11: 55-63.
  - IREF - Iregren, A. 1982. Effects on psychological test performance of workers exposed to a single solvent (toluene) - a comparison with effects of

- exposure to a mixture of organic solvents. *Neurobehav. Toxicol. Teratol.* 4(6): 695-701.
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- IREF - Pryor, G.T., C.S. Rebert, J. Dickinson and E.M. Feeney. 1984. Factors affecting toluene-induced ototoxicity in rats. *Neurobehav. Toxicol. Teratol.* 6: 223-238.
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- IREF - U.S. EPA. 1984. Health Effects Assessment for Toluene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Emergency and Remedial Response, Washington, DC. EPA-600/X-84-188.
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- IREF - Yin, S., G. Li, Y. Hu et al. 1987. Symptoms and signs of workers exposed to benzene, toluene or the combination. *Ind. Health.* 25(3): 113-130.
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Option? CAS/79016

File: 4 Count: 1

Option? TYPE 4/2

File 4; Entry 1; Accession No. 1199

(CAS) CAS Registry Number: 79-01-6

(MAT) Material Name: Trichloroethylene

(SYN) Synonyms:

ACETYLENE TRICHLORIDE;  
ALGYLEN;  
ANAMENTH;  
BENZINOL;  
BLACOSOLV;  
BLANCOSOLV;  
CECOLENE;  
CHLORILEN;  
1-CHLORO-2,2-DICHLOROETHYLENE;  
CHLORYLEA;  
CHLORYLEN;  
CHORYLEN;  
CIRCOSOLV;  
CRAWHASPOL;  
DENSINFLUAT;  
1,1-DICHLORO-2-CHLOROETHYLENE;  
DOW-TRI;  
DUKERON;  
ETHINYL TRICHLORIDE;  
ETHYLENE TRICHLORIDE;  
ETHYLENE, TRICHLORO-;  
FLECK-FLIP;  
FLOCK FLIP;  
FLUATE;  
GEMALGENE;  
GERMALGENE;  
LANADIN;  
LETHURIN;  
NARCOGEN;  
NARKOGEN;  
NARKOSOID;  
NCI-C04546;  
NIALK;  
PERM-A-CHLOR;  
PERM-A-CLOR;  
PETZINOL;  
PHILEX;  
RCRA WASTE NUMBER U228;  
TCE;  
THRETHYLEN;  
THRETHYLENE;

TRETHYLENE;  
 TRI;  
 TRIAD;  
 TRIAL;  
 TRIASOL;  
 TRICHLOORETHEEN;  
 TRICHLOORETHYLEEN, TRI;  
 TRICHLORAETHEN;  
 TRICHLORAETHYLEN, TRI;  
 TRICHLORAN;  
 TRICHLOREN;  
 TRICHLORETHENE;  
 TRICHLORETHYLENE;  
 TRICHLORETHYLENE, TRI;  
 TRICHLOROETHENE;  
 Trichloroethylene;  
 1,1,2-TRICHLOROETHYLENE;  
 1,2,2-TRICHLOROETHYLENE;  
 TRI-CLENE;  
 TRICLORETENE;  
 TRICLOROETILENE;  
 TRIELENE;  
 TRIELIN;  
 TRIELINA;  
 TRIKLONE;  
 TRILEN;  
 TRILENE;  
 TRILINE;  
 TRIMAR;  
 TRIOL;  
 TRI-PLUS;  
 TRI-PLUS M;  
 UN 1710;  
 VESTROL;  
 VITRAN;  
 WESTROSOL

(UPD) Update Date: 06-01-90

(EFF) Effective Date: 07-01-91

(STAT) Status:  
 STATUS OF DATA FOR Trichloroethylene

File On-Line 03-31-87

Category (section) -----	Status -----	Last Revised -----
Oral RfD Assessment (I.A.)	pending	
Inhalation RfC Assessment (I.B.)	pending	
Carcinogenicity Assessment (II.)	wichdrawn	07-01-89

Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	on-line	06-01-90
Supplementary Data (V.)	no data	

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(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

A risk assessment for this substance/agent is under review by an EPA work group.

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

A risk assessment for this substance/agent is under review by an EPA work group.

(CAR) Carcinogenicity Assessment:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

The carcinogen assessment summary for this substance has been withdrawn following further review. A new carcinogen summary is in preparation by the CRAVE Work Group.

Contact: Rita S. Schoeny / ORD / FTS/684-7544 or 513/569-7544

(REGS) Regulations:

III. HEALTH HAZARD ASSESSMENTS FOR VARIED EXPOSURE DURATIONS

IV. U.S. EPA REGULATORY ACTIONS

IV.A. CLEAN AIR ACT (CAA)

IV.A.1. CAA REGULATORY DECISION

Action -- Intent to list under Section 112

Considers technological or economic feasibility? -- NO

Discussion -- Trichloroethylene (TCE) is a probable human carcinogen (EPA Group B2) and according to EPA's preliminary risk assessment from ambient air

exposures, public health risks are significant (4.1 cancer cases/year and maximum lifetime individual risks of  $9.4 \times 10^{-5}$ ). Thus, EPA indicated that it

intends to add TCE to the list of hazardous air pollutants for which it intends to establish emission standards under section 112(b)(1)(A) of the Clean Air Act. The EPA will decide whether to add TCE to the list only after

studying possible techniques that might be used to control emissions and further assessing the public health risks. The EPA will add TCE to the list

if emissions standards are warranted.

Reference -- 50 FR 52422 (12/23/85)

EPA Contact -- Emissions Standards Division, OAQPS  
(919)541-5571 / FTS 629-5571

#### IV.B. SAFE DRINKING WATER ACT (SDWA)

##### IV.B.1. MAXIMUM CONTAMINANT LEVEL GOAL (MCLG) for Drinking Water

Value (status) -- 0 mg/L (Proposed, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- An MCLG of 0 mg/L for trichloroethylene is proposed based on carcinogenic effects. Significant increases in the incidence of liver tumors have been reported in B6C3F1 mice of both sexes. Malignant lymphomas and pulmonary adenocarcinomas were also reported in mice. EPA has classified trichloroethylene in Group B2: sufficient evidence in animals and inadequate evidence in humans.

Reference -- 50 FR 46880 Part III (11/13/85)

EPA Contact -- Criteria and Standards Division, ODW /  
(202)382-7571 / FTS 382-7571; or Drinking Water Hotline / (800)426-4791

##### IV.B.2. MAXIMUM CONTAMINANT LEVEL (MCL) for Drinking Water

Value (status) -- 5 ug/L (Final, 1987)

Considers technological or economic feasibility? -- YES

Discussion --

Reference -- 52 FR 35690

EPA Contact -- Criteria and Standards Division, ODW /  
(202)382-7571 / FTS 382-7571; or Drinking Water Hotline / (800)426-4791

IV.C. CLEAN WATER ACT (CWA)

IV.C.1. AMBIENT WATER QUALITY CRITERIA, Human Health

Water and Fish Consumption --  $2.7E+0$  ug/L

Fish Consumption Only --  $8.07E+1$  ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero.

However, zero may not be attainable at this time, so the recommended criteria represents a E-6 estimated incremental increase of cancer risk over a lifetime.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division, OWRS  
(202)475-7315 / FTS 475-7315

IV.C.2. AMBIENT WATER QUALITY CRITERIA, Aquatic Organisms

Freshwater:

Acute LEC --  $4.5E+4$  ug/L

Chronic LEC -- None

Marine:

Acute LEC --  $2.0E+3$  ug/L

Chronic LEC -- None

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LECs are given when the minimum data required to derive water quality criteria are not available.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division, OWRS  
(202)475-7315 / FTS 475-7315

IV.F. RESOURCE CONSERVATION AND RECOVERY ACT (RCRA)

IV.F.1. RCRA APPENDIX IX, for Ground Water Monitoring

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

IV.G. SUPERFUND (CERCLA)

IV.G.1. REPORTABLE QUANTITY (RQ) for Release into the Environment

Value (status) -- 100 pounds (Proposed, 1987)

Considers technological or economic feasibility? -- NO

Discussion -- The proposed RQ for trichloroethylene is 100 pounds, based on potential carcinogenic . The available data indicate a hazard ranking of low, based on a potency factor of 0.070 (mg/kg/day)-1 and weight-of-evidence classification B2, which corresponds to an RQ of 100 pounds.

Reference -- 52 FR 8140 (03/16/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

Option? TYPE 2/2

File 2; Entry 1; Accession No. 1120

(CAS) CAS Registry Number: 75-69-4

(MAT) Material Name: Trichlorofluoromethane



(SYN) Synonyms:

ALGOFRENE TYPE 1;  
ARCTON 9;  
ELECTRO-CF 11;  
ESKIMON 11;  
F 11;  
FC 11;  
FLUOROCARBON NO. 11;  
FLUOROTRICHLOROMETHANE;  
FLUOROTROJCHLOROMETAN;  
FREON 11;  
FREON 11A;  
FREON 11B;  
FREON HE;  
FREON MF;  
FRIGEN 11;  
GENETRON 11;  
HALOCARBON 11;  
ISCEON 131;  
ISOTRON 11;  
LEDON 11;  
MONOFLUOROTRICHLOROMETHANE;  
NCI-C04637;  
RCRA WASTE NUMBER U121;  
Trichlorofluoromethane;  
TRICHLOROMONOFUOROMETHANE;  
UCON FLUROCARBON 11;  
UCON REFRIGERANT 11

(UPD) Update Date: 08-01-90

(EFF) Effective Date: 07-01-91

(STAT) Status:

STATUS OF DATA FOR Trichlorofluoromethane

File On-Line 01-31-87

Category (section)

Status

Last Revised

.....  
Oral RfD Assessment (I.A.)

.....  
on-line

.....  
08-01-90

Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	no data	
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	on-line	08-01-90
Supplementary Data (V.)	no data	

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(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
-----	-----	-----	---	-----
Survival and histo- pathology	NOAEL: none	1000	1	3E-1 mg/kg/day
Cancer Bioassay Studies in Rats and Mice	LOAEL: 488 mg/kg/day converted to 349 mg/ kg/day			
NCI, 1978				

---

\*Conversion Factors: 5 days/7 days; thus, 488 mg/kg/day x 5 days/7 days =  
349 mg/kg/day

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

NCI (National Cancer Institute). 1978. Bioassay of trichlorofluoromethane for possible carcinogenicity. Report. No. 106, PHS/NIH, DHEW Publ. No. 78-1356.

The NCI bioassay was performed on rats and mice exposed to various doses of trichloromonofluoromethane by gavage over a period of 78 weeks (50 animals/species/sex/dose for each of two doses with 20 animals/species/sex for each of two control groups). A statistically significant positive association

between increased dosage and accelerated mortality by the Tarone test in male and female rats and female mice were observed. In treated rats of both sexes there were also elevated incidences of pleuritis and pericarditis not seen in controls. Inhalation studies which employed multispecies exposures to higher levels of the compound than used by NCI (Leuschner et al., 1983; Colman et al., 1981; Hansen et al., 1984) reported no adverse clinical/pathologic signs of toxicity due to subchronic or short-term exposures.

The LOAEL of 488 mg/kg/day (based on mortality in rats) was converted to 349 mg/kg/day on a 7-day exposure basis.

#### I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF = 1000. An uncertainty factor of 1000 (10 for LOAEL, 10 for species conversion, and 10 for sensitive human population), results in an ADI of 0.3 mg/kg/day.

MF = 1.

#### I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

None.

#### I.A.5. CONFIDENCE IN THE ORAL RfD

Study: Medium  
Data Base: Medium  
RfD: Medium

The chosen study is given a medium confidence rating because large numbers of animals/sex were tested in two doses for chronic exposures, but the study did not establish a NOEL. The data base is given a medium confidence rating because of the support of chronic data, but the lack of reproductive data. Medium confidence in the RfD follows.

#### I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

The only U.S. EPA documentation at present is on IRIS.

Agency RfD Work Group Review: 05/20/85, 05/31/85

Verification Date: 05/31/85

I.A.7. EPA CONTACTS (ORAL RFD)

Christopher T. DeRosa / ORD -- (513)569-7534 / FTS 684-7534

Michael L. Dourson / ORD -- (513)569-7544 / FTS 684-7544

(CAR) Carcinogenicity Assessment:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

This substance/agent has not been evaluated by the U.S. EPA for evidence of human carcinogenic potential.

(REGS) Regulations:

III. HEALTH HAZARD ASSESSMENTS FOR VARIED EXPOSURE DURATIONS

IV. U.S. EPA REGULATORY ACTIONS

IV.A. CLEAN AIR ACT (CAA)

No data available

IV.C. CLEAN WATER ACT (CWA)

IV.C.1. AMBIENT WATER QUALITY CRITERIA, Human Health

Water and Fish Consumption:  $1.9E-1$  ug/L

Fish Consumption Only:  $1.57E+1$  ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The WQC of  $0.19$  ug/L represents a cancer risk level of  $1E-6$ , based on consumption of contaminated organisms and water. A WQC of  $15.7$  ug/L (cancer risk level of  $1E-6$ ) has also been established based on consumption of contaminated organisms alone. The criteria are based on halomethanes as a class.

Reference -- 45 FR 79318 (11/28/80)

E: Contact -- Criteria and Standards Division, OWRS  
(202)475-7315 / FTS 475-7315

IV.C.2. AMBIENT WATER QUALITY CRITERIA, Aquatic Organisms

Freshwater:

Acute LEC -- 1.1E+4 ug/L  
Chronic -- None

Marine:

Acute LEC -- 1.2E+4 ug/L  
Chronic LEC -- 6.4E+3 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LECs are given when the minimum data required to derive water quality criteria are not available.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division, OWRS  
(202)475-7315 / FTS 475-7315

IV.E. TOXIC SUBSTANCES CONTROL ACT (TSCA)

IV.E.1. TSCA, SECTION 6

Status -- Final (1978)

Discussion -- 40 CFR Part 762.1 prohibits the manufacture, processing and distribution in commerce of fully halogenated chlorofluoralkanes for those aerosol propellant uses which are subject to TSCA, (with exception of listed exemptions) requires submission of annual reports and lists exemptions from the prohibition.

Reference -- 40 CFR Part 762 - Fully Halogenated Chlorofluoralkanes

EPA Contact -- Chemical Control Division, OTS / (202)382-3749 / FTS 382-3749

IV.F. RESOURCE CONSERVATION AND RECOVERY ACT (RCRA)

IV.F.1. RCRA APPENDIX IX, for Ground Water Monitoring

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

IV.G. SUPERFUND (CERCLA)

IV.G.1. REPORTABLE QUANTITY (RQ) for Release into the Environment

Value (status) -- 5000 pounds (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- No data have been found that permit the ranking of this hazardous substance. The available data for the acute hazards may lie above the upper limit for the 5000-pound RQ, but since it is a designated hazardous substance, the largest assignable RQ is 5000 pounds.

Reference -- 50 FR 13456 (04/04/85)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

File 7; Entry 1; Accession No. 1125

(CAS) CAS Registry Number: 1314-62-1

(MAT) Material Name: Vanadium pentoxide

(SYN) Synonyms:

CI 77938;  
Divanadium Pentaoxide;  
Divanadium Pentoxide;  
Vanadic Anhydride;  
Vanadium Oxide;  
Vanadium Pentaoxide;  
Vanadium Pentoxide

(UPD) Update Date: 06-30-88

(EFF) Effective Date: 10-01-91

(STAT) Status:

STATUS OF DATA FOR Vanadium pentoxide

File On-Line 01-31-87

Category (section)	Status	Last Revised
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Oral RfD Assessment (I.A.)	on-line	06-30-88
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	message	06-30-88
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	on-line	03-01-88
Supplementary Data (V.)	on-line	01-31-87

(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
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Decreased hair cystine	NOAEL: 17.85 ppm converted to 0.89 mg/kg/day	100	1	9E-3 mg/kg/day
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Rat Chronic Oral Study	LOAEL: none
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Stokinger et al., 1953

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\*Conversion Factor: Adult rat food consumption assumed to be 5% bw/day.

#### I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Stokinger, H.E., W.D. Wagner, J.T. Mountain, F.R. Stacksill, O.J. Dobrogorski and R.G. Keenan. 1953. Unpublished results. Division of Occupational Health, Cincinnati, OH. (Cited in Patty's Industrial Hygiene and Toxicology, 3rd ed., 1981)

In this chronic study, an unspecified number of rats were exposed to dietary levels of 10 or 100 ppm vanadium (about 17.9 or 179 ppm vanadium pentoxide) for 2.5 years. The results of this unpublished study were summarized by Stokinger et al. (1981). The criteria used to evaluate vanadium toxicity were growth rate, survival, and hair cystine content. The only significant change reported was a decrease in the amount of cystine in the hair of animals ingesting vanadium.

Of the subchronic and chronic animal studies available, the lower dose level (17.9 ppm vanadium pentoxide) reported in the Stokinger et al. (1953) study is the highest oral NOAEL upon which an RfD can be derived. An oral RfD of 0.009 mg/kg/day (0.62 mg/day for a 70-kg person) can be calculated by assuming that rats eat food equivalent to 5% of their body weight and by applying an uncertainty factor of 100.

#### I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF = 100. An uncertainty factor of 100 was applied, 10 for interspecies extrapolation and a factor of 10 to provide added protection for unusually sensitive individuals.

MF = 1.

#### I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

In a subchronic feeding study (Mountain et al., 1953), groups of five male Wistar rats were fed vanadium pentoxide at levels of 0, 25, or 50 ppm for 35 days, after which dietary levels of vanadium were increased to 100 and 150 ppm and continued for 68 days. There was a decrease in the amount of cystine in the hair of the high-dosed (50-150 ppm or 2.5-7.5 mg/kg/day, based on food consumption of 5% bw) rats. A significant decrease was also reported in erythrocyte and hemoglobin levels of the high-dosed rats. In an abstract of a subchronic inhalation study (Sugira, 1978), mice and rats exposed to 1 to 3 mg/cu.m vanadium pentoxide for 3 months, 6 hours/day developed histopathologic changes in their lungs and had a decrease in growth rate. Adverse effects were not detected in either species similarly exposed at 0.1 to 0.4



mg/cu.m.

Although several epidemiologic studies have been conducted on factory workers exposed to vanadium pentoxide for several years, the air concentration levels of vanadium pentoxide were measured only at scattered intervals, making it impossible to determine a minimum effective dose. Also, in cases of humans exposed to relatively high atmospheric concentrations of vanadium pentoxide for short periods of time, all individuals developed respiratory symptoms that usually subsided within 7-14 days.

#### I.A.5. CONFIDENCE IN THE ORAL RfD

Study: Low  
Data Base: Low  
RfD: Low

Because of the lack of details in the reference study and the scarcity of data available on vanadium pentoxide, low confidence is assigned to both the study and the data base. Low confidence in the RfD follows.

#### I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

The only U.S. EPA documentation at present is on IRIS.

Agency Work Group Review: 02/26/86

Verification Date: 02/26/86

#### I.A.7. EPA CONTACTS (ORAL RfD)

Christopher T. DeRosa / ORD -- (513)569-7534 / FTS 684-7534

Michael L. Dourson / ORD -- (513)569-7544 / FTS 684-7544

(CAR) Carcinogenicity Assessment:

#### II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

The NTP (1985) has approved vanadium pentoxide for carcinogenicity testing; however, the route of administration has not been determined (i.e., oral, inhalation).

(REGS) Regulations:

#### III. HEALTH HAZARD ASSESSMENTS FOR VARIED EXPOSURE DURATIONS

#### IV. U.S. EPA REGULATORY ACTIONS

##### IV.A. CLEAN AIR ACT (CAA)

No data available

IV.F. RESOURCE CONSERVATION AND RECOVERY ACT (RCRA)  
IV.F.1. RCRA APPENDIX IX, for Ground Water Monitoring

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

IV.G. SUPERFUND (CERCLA)

IV.G.1. REPORTABLE QUANTITY (RQ) for Release into the Environment

Value (status) -- 1000 pounds (Final, 1986)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ is based on aquatic toxicity (as established under Section 311(b)(4) of the Clean Water Act), chronic toxicity and acute toxicity. The available data indicate that the aquatic 96-hour Median Threshold Limit for vanadium pentoxide is between 10 and 100 ppm. RQ assignments based on chronic toxicity reflect two primary attributes of the hazardous substance, the minimum effective dose (MED) levels for chronic exposure (mg/day for 70-kg man) and the type of effect (liver necrosis, teratogenicity, etc. The composite score of these two attributes for vanadium pentoxide is between 6 and 20, corresponding to a chronic toxicity RQ of 1000 pounds. In addition, the oral LD50 for rats is between 10 and 100 mg/kg and the inhalation LC10 for rats is between 40 and 400 ppm, also a 1000-pound RQ.

Reference -- 51 FR 34534 (09/29/86)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

(PROP) Physical-Chemical Properties:

V.B. PHYSICAL-CHEMICAL PROPERTIES

Chemical Formula -- V2O5

Molecular Weight -- 181.90

Boiling Point -- 3182F, 1750C (decomposition)

Specific Gravity (H2O-1) -- 3.357 at 18C

Vapor Pressure (mmHg) -- Approximately 0 at 20C, 68F

Melting Point -- 1274F, 690C

Vapor Density (AIR-1) -- Not Found

Evaporation Rate (Butyl acetate-1) -- Not Found

Solubility in Water -- 1 g in 125 mL

Flash Point [Method Used] -- Not Found

Flammable Limits -- Not Flammable

Appearance and Odor -- Vanadium pentoxide exists as a yellow-orange powder, dark gray flakes, or yellow to rust brown crystals (NIOSH/OSHA, 1981; Merck, 1983). It is odorless (CHRIS, 1978)

Conditions or Materials to Avoid -- Avoid chlorine trifluoride; lithium; peroxyformic acid; and calcium, sulfur, water complexes (Sax, 1984, p. 2718)

Hazardous Decomposition or Byproducts -- When heated to decomposition, it emits acrid smoke and fumes of vanadium oxides (Sax, 1984, p. 2718).

Use -- Vanadium pentoxide is used as a catalyst in the oxidation of sulfur dioxide to sulfur trioxide, alcohol to acetaldehyde, etc.; for the manufacture of yellow glass; inhibiting ultraviolet light transmission in glass; as a depolarizer; as a developer in photography; in form of ammonium vanadate as mordant in dyeing and printing fabrics and in manufacture of aniline black (Merck, 1983, p. 1418).

Option? CAS/1330207  
File: 12 Count: 1

Option? TYPE 12/2

File 12; Entry 1; Accession No. 1270

(CAS) CAS Registry Number: 1330-20-7

(MAT) Material Name: Xylenes

(SYN) Synonyms:  
dimethylbenzene;  
1,2-dimethylbenzene;  
1,3-dimethylbenzene;  
1,4-dimethylbenzene;  
mixed xylenes;  
m-xylene;  
meta-xylene;  
o-xylene;  
ortho-xylene;  
p-xylene;  
para-xylene;  
Xylenes

(UPD) Update Date: 03-01-91

(EFF) Effective Date: 07-01-91

(STAT) Status:  
STATUS OF DATA FOR Xylenes

File On-Line 09-30-87

Category (section)	Status	Last Revised
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Oral RfD Assessment (I.A.)	on-line	09-30-87
Inhalation RfC Assessment (I.B.)	pending	
Carcinogenicity Assessment (II.)	on-line	03-01-91
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	on-line	03-01-91
Supplementary Data (V.)	no data	

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(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Hyperactivity, decreased body weight and increased mortality (males)	NOAEL: 250 mg/kg/day (converted to 179 mg/kg/day)	100	1	2E+0 mg/kg/day
Chronic Rat Gavage Study	FEL: 500 mg/kg/day (converted to 357 mg/kg/day)			
NTP, 1986				

\*Conversion Factors: Dose adjusted for gavage schedule (5\days/week).

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

NTP (National Toxicology Program). 1986. NTP Technical Report on the Toxicology and Carcinogenesis of Xylenes (mixed) (60.2% m-xylene, 13.6% p-xylene, 17.0 ethylbenzene and 9.1% O-xylene) (CAS No. 1330-20-7) in F344/N rats and B6C3F1 mice (gavage studies). U.S. DHHS, PHS, NIH, NTP, Research Triangle Park, NC. NTP TR 327, NIH Publ. No. 86-2583.

Groups of 50 male and 50 female Fischer 344 rats and 50 male and 50 female B6C3F1 mice were given gavage doses of 0, 250, or 500 mg/kg/day (rats) and 0, 500, or 1000 mg/kg/day (mice) for 5 days/week for 103 weeks. The animals were observed for clinical signs of toxicity, body weight gain, and mortality. All animals that died or were killed at sacrifice were given gross necropsy and comprehensive histologic examinations. There was a dose-related increased mortality in male rats, and the increase was significantly greater in the high-dose group compared with controls. Although increased mortality was observed at 250 mg/kg/day, the increase was not significant. Although many of the early deaths were caused by gavage error, NTP (1986) did not rule out the possibility that the rats were resisting gavage dosing because of the behavioral effects of xylene. Mice given the high dose exhibited hyperactivity, a manifestation of CNS toxicity. There were no compound-related

histopathologic lesions in any of the treated rats or mice. Therefore, the high dose is a FEL and the low dose a NOAEL.

#### I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF = 100. An uncertainty factor of 100 was chosen: 10 for species-to-species extrapolation and 10 to protect sensitive individuals.

MF = 1.

#### I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

U.S. EPA (1984) reported an RfD of 0.01 mg/kg/day, based on a rat dietary NOAEL of 200 ppm or 10 mg/kg/day as defined by Bowers et al. (1982) in a 6-month study. This NOAEL was divided by an uncertainty factor of 1000. U.S. EPA (1985, 1986) noted that this study used aged rats, loss of xylene from volatilization was not controlled, only one exposure level was used, and histopathologic examination was incomplete. An RfD of 4.31 mg/day (about 0.06 mg/kg/day) based on an inhalation study (Jenkins et al., 1970) using rats, guinea pigs, monkeys, and dogs exposed to o-xylene at 3358 mg/cu.m, 8 hours/day, 5 days/week for 6 weeks or at 337 mg/cu.m continuously for 90 days was derived by U.S. EPA (1985). Deaths in rats and monkeys, and tremors in dogs occurred at the highest dose, whereas no effects were observed in the 337 mg/cu.m continuous exposure group. The RfD based on the NTP (1986) study is preferable because it is based on a chronic exposure in two species by a relevant route of administration, and comprehensive histology was performed. Xylene is fetotoxic and teratogenic in mice at high oral doses (Nawrot and Staples, 1981; Marks et al., 1982), but the RfD as calculated should be protective of these effects.

#### I.A.5. CONFIDENCE IN THE ORAL RfD

Study: Medium  
Data Base: Medium  
RfD: Medium

The NTP (1986) study was given a medium confidence level because it was a well-designed study in which adequately sized groups of two species were tested over a substantial portion of their lifespan, comprehensive histology was performed, and a NOAEL was defined; but clinical chemistries, blood enzymes, and urinalysis were not performed. The data base was given a medium confidence level because, although supporting data exist for mice and

teratogenicity and fetotoxicity data are available with positive results at high oral doses, a LOAEL for chronic oral exposure has not been defined. Medium confidence in the RfD follows.

#### I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

U.S. EPA. 1986. Health and Environmental Effects Profile for Xylenes (o-, m-, p-). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH and the Environmental Criteria and Assessment Office, Research Triangle Park, NC for the Office of Solid Waste and Emergency Response and the Office of Air Quality Planning and Standards, Office of Air and Radiation, Washington, DC. Limited peer review and extensive agency-wide review, 1986.

U.S. EPA. 1985. Drinking Water Criteria Document For Xylenes. Prepared by Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.

Extensive peer review agency-wide review.

U.S. EPA. 1984. Health Effects Assessment for Xylene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Emergency and Remedial Response, Washington, DC.

ECAO internal review and limited agency review.

Agency RfD Work Group Review: 12/05/85, 03/19/87

Verification Date: 03/19/87

#### I.A.7. EPA CONTACTS (ORAL RfD)

Harlal Choudhury / ORD -- (513)569-7536 / FTS 684-7536

Christopher T. DeRosa / ORD -- (513)569-7534 / FTS 684-7534

#### I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

A risk assessment for this substance/agent is under review by an EPA work group.

(CAR) Carcinogenicity Assessment:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity.

Basis -- Orally administered technical xylene mixtures did not result in significant increases in incidences in tumor responses in rats or mice of both sexes.

II.A.2. HUMAN CARCINOGENICITY DATA

None.

II.A.3. ANIMAL CARCINOGENICITY DATA

Inadequate. In an NTP (1986) study, 50 male and 50 female F344/N rats were treated by gavage with mixed xylenes in corn oil (60% m-xylene, 14% p-xylene, 9% o-xylene and 17% ethylbenzene) at dosages of 0, 250 or 500 mg/kg/day, 5 days/week for 103 weeks. Similarly, 50 male and 50 female B6C3F1 mice were treated with the same xylene mixture at dosages of 0, 500 or 1000 mg/kg/day. Animals were killed and examined histologically when moribund or after 104-105 weeks. An apparent dose-related increased mortality was observed in male rats, but this difference was statistically significant for the high dose group, only. No other differences in survival between dosage groups of either sex were observed. Interstitial cell tumors of the testes could not be attributed to administration of the test compound observed in male rats (43/50 control, 38/50 low-dose and 41/49 high-dose). NTP (1986) reported that there were no significant changes in the incidence of neoplastic or nonneoplastic lesions in either the rats or mice that could be considered related to the mixed xylene treatment, and concluded that under the conditions of these 2-year gavage studies, there was "no evidence of carcinogenicity" of xylene (mixed) for rats or mice of either sex at any dosage tested.

Maltoni et al. (1985), in a limited study, reported higher incidences (compared with controls) of malignant tumors in male and female Sprague-Dawley rats treated by gavage with xylene in olive oil at 500 mg/kg/day, 4 or 5 days/week for 104 weeks. This study did not report survival rates or specific tumor types; therefore, the results cannot be interpreted.

Berenblum (1941) reported that "undiluted" xylene applied at weekly intervals produced one tumor-bearing animal out of 40 after 25 weeks in skin-painting experiments in mice. No control groups were described. Pound



(1970) reported negative results in initiation-promotion experiments with xylene as the initiator and croton oil as the promotor.

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

The frequency of sister chromatid exchanges and chromosomal aberrations were nearly identical between a group of 17 paint industry workers exposed to xylene and their respective referents (Haglund et al., 1980). In vitro, xylene caused no increase in the number of sister chromatid exchanges in human lymphocytes (Gerner-Smidt and Friedrich, 1978). Studies indicate that xylene isomers, technical grade xylene or mixed xylene are not mutagenic in tests with *Salmonella typhimurium* (Florin et al., 1980; NTP, 1986; Bos et al., 1981) nor in mutant reversion assays with *Escherichia coli* (McCarroll et al., 1981). Technical grade xylene, but not o- and m-xylene, was weakly mutagenic in *Drosophila* recessive lethal tests. Chromosomal aberrations were not increased in bone marrow cells of rats exposed to xylenes by inhalation (Donner et al., 1980).

#### II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

##### II.D.1. EPA DOCUMENTATION

U.S. EPA. 1987. Drinking Water Criteria Document for Xylene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. ECAO-CIN-416. Final.

##### II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The Drinking Water Criteria Document for Xylene has received Agency and external review.

Agency Work Group Review: 12/02/87

Verification Date: 12/02/87

##### II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Bruce Mintz / ODW -- (202)475-9569 / FTS 475-9569

W. Bruce Peirano / ORD -- (513)569-7540 / FTS 684-7540

(REGS) Regulations:

III. HEALTH HAZARD ASSESSMENTS FOR VARIED EXPOSURE DURATIONS

IV. U.S. EPA REGULATORY ACTIONS

IV.A. CLEAN AIR ACT (CAA)

No data available

IV.B. SAFE DRINKING WATER ACT (SDWA)

IV.B.1. MAXIMUM CONTAMINANT LEVEL GOAL (MCLG) for Drinking Water

Value (status) -- 0.44 mg/L (Proposed, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- An MCLG of 0.44 mg/L for xylene is proposed based upon a DWEL of 2.2 mg/L and an assumed drinking water contribution of 20%. A DWEL (provisional) of 2.2 mg/L was calculated from a NOAEL of 337 mg/cu.m (only dose tested) for body weight, hematology and histopathologic effects in rats, guinea pigs, monkeys and dogs in a 90-day inhalation study (Jenkins, 1970).

An uncertainty factor of 1000 was applied and human water consumption of 2 L/day was assumed.

Reference -- 50 FR 46936 Part IV (11/13/85)

EPA Contact -- Yogendra Patel / Criteria and Standards Division, ODW / (202)382-7571 / FTS 382-7571; or Drinking Water Hotline / (800)426-4791

IV.F. RESOURCE CONSERVATION AND RECOVERY ACT (RCRA)

IV.F.1. RCRA APPENDIX IX, for Ground Water Monitoring

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

IV.G. SUPERFUND (CERCLA)

IV.G.1. REPORTABLE QUANTITY (RQ) for Release into the Environment

Value (status) -- 1000 pounds (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ is based on ignitability and aquatic toxicity as established for xylene under Section 311(b)(4) of the Clean Water Act (40 CFR 117.3). The available data indicate the aquatic 96-hour Median Threshold Limit for xylene is between 10 and 100 ppm, corresponding to an RQ of 1000 pounds. The ignitibility RQ of 1000 pounds is based on a flash point of 81 to 90F.

Reference -- 50 FR 13456 (04/04/85)

EPA Contact -- RCRA/Superfund Hotline  
(800)424-9346 / (202)382-3000 / FTS 382-3000

Option? TYPE 3/2

File 3; Entry 1; Accession No.

(CAS) CAS Registry Number: 7440-66-6

(MAT) Material Name: Zinc and Compounds

(SYN) Synonyms:

Zinc;  
Asarco L 15;  
Blue powder;  
Cinc [Spanish];  
EMANAY ZINC DUST;  
GRANULAR ZINC;  
HSDB 1344;  
JASAD;  
Lead refinery vacuum zinc;  
Merrillite;  
UN 1436;  
Zinc;  
ZINC DUST;  
ZINC POWDER;  
ZINC, ashes;  
ZINC, powder or dust, non-pyrophoric;  
ZINC, powder or dust, pyrophoric

(UPD) Update Date: 02-01-91

(EFF) Effective Date: 07-01-91

(STAT) Status:

STATUS OF DATA FOR Zinc and Compounds

File On-Line 02-01-91

Category (section)	Status	Last
Revised		
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-----		
Oral RfD Assessment (I.A.)	pending	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	
02-01-91		
Drinking Water Health Advisories (III.A.)	no data	

U.S. EPA Regulatory Actions (IV.)

no data

Supplementary Data (V.)

no data

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(HAZ) Chronic Health Hazards, Noncarcinogenic:

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

A risk assessment for this substance/agent is under review by an EPA work group.

(CAR) Carcinogenicity Assessment:

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity

Basis -- Based on inadequate evidence in humans and animals.

II.A.2. HUMAN CARCINOGENICITY DATA

Inadequate. There are no reports on the possible carcinogenicity of zinc and compounds per se in humans. Case studies have been used to evaluate the effects of zinc administered for therapeutic reasons. There are reports which compare zinc levels in normal and cancerous tissue. Studies of occupational exposure to zinc compounds have also been conducted, but have limited value because they do not correlate exposure with cancer risk.

Case reports of chronic therapeutic exposure for approximately 2 years of two patients, a 59-year-old female and a 26-year-old homozygous sickle-cell male, to 100-150 mg/day zinc as zinc sulfate or zinc acetate,

respectively,  
have reported a profound anemia associated with hypoceruloplasminemia and hypocupremia (Porter et al., 1977; Prasad et al., 1978). The conditions were corrected by copper supplementation and, in one case, withdrawal of zinc.

Habib et al. (1976) reported that average zinc concentrations in normal and hypertrophic prostate tissues were similar, approximately 6.8  $\mu\text{mol/g}$ , but the average zinc concentration was lower in carcinomatous prostate tissues (2.6  $\mu\text{mol/g}$ ). These tissue samples were obtained as follows: normal prostate tissues were obtained at autopsy from 9 men 25-58 years old (average age 36); and both hyperplastic and carcinomatous prostate tissues were obtained from the biopsies of 23 men 58-87 years old (average age 70) and from 9 men 64-91 years old (average age 73), respectively. Several other studies have also shown lower average zinc concentrations in cancerous vs. normal or hypotrophic prostate tissue (U.S. EPA, 1987). NRC (1978) and U.S. EPA (1987) have reviewed other studies which have noted both high and low zinc levels in other cancerous and noncancerous tissues with no definite pattern. From these studies it could not be concluded whether zinc was a carcinogen.

Several occupational studies have been conducted on workers exposed to zinc compounds (Batchelor et al., 1926; Chmielewski et al., 1974a,b; Bobrishchev-Pushkin et al., 1977). No increase in the incidence of cancer was noted; however, the studies were designed to evaluate other endpoints and did not specifically address cancer. Other symptoms such as slight leukocytosis, occurrences of metal fume fever, respiratory disease and hypocalcemia were some of the findings noted in exposed workers. Batchelor et al. (1926) extensively investigated workers exposed to zinc in a smelter. A total of 24 workers whose exposure ranged from 2-35.5 years were selected. In most work areas the mean zinc concentrations were generally below 35

mg/cu.m, except in the zinc dust plant where concentrations of up to 130 mg/cu.m were measured.

The average level of zinc in whole blood of the 24 exposed workers was 458

ug/100 mL, compared with 387 ug/100 mL in 10 control measurements. No

information was given about the control subjects. Klucik and Koprda (1979)

found that exposure levels to zinc oxide dust in a zinc oxide factory were on

average 0.5 mg/cu.m for zinc melters and 2.44-7.15 mg/cu.m for zinc oxide

packers; it was not indicated how these values were obtained. Chmielewski et

al. (1974a,b) examined a group of workers who were exposed to zinc oxide in a

shipyard; this included 20 ship smiths, 20 electric welders, 20 ship's

pipeline fitters, and 20 zincifying workers. High concentrations of zinc

oxide were found at the stands of the electric welders, who worked in

containers (maximum 58 mg/cu.m, mean 18 mg/cu.m), and the ship smiths, who

worked in a superstructure (maximum 50 mg/cu.m, mean 12 mg/cu.m). These

workers were also exposed to other hazardous compounds, such as nitrogen

oxides. Bobrishchev-Pushkin et al. (1977) studied 1018 workers in the casting

shops of three copper alloy production facilities in the USSR. Four hundred

and fifty-one workers from the rolling shops were used as controls. The

average level of zinc oxide exposure in the casting shop was 2.1 mg/cu.m

(range of 0.2-5.1 mg/cu.m), well below the USSR's maximally allowable

concentration of 6 mg/cu.m. Workers were also exposed to other metals such as

copper, lead and nickel.

#### II.A.3. ANIMAL CARCINOGENICITY DATA

Inadequate. In a 1-year study, an unspecified number of newborn Chester

Beatty stock mice (sex not reported) were administered 0, 1000, or 5000 ppm

zinc (approximately 0, 170, or 850 mg/kg/day) as zinc sulfate in drinking

water (Walters and Roe, 1965). A separate group of mice received zinc oleate

in the diet at an initial dose of 5000 ppm zinc; this dose was reduced to 2500 ppm after 3 months and to 1250 ppm after an additional 3 months because of mortality due to anemia. An epidemic of ectromelia caused the deaths of several mice during the first 8 weeks; consequently, additional control and test-diet groups were established. There was no difference in body weight gain between control and treated groups, except the dietary zinc group which became anemic. Survival was not reported in treated compared with control groups.

An apparent increase in the incidence of hepatomas was observed in treated mice surviving for 45 weeks or longer relative to controls (original and replacement mice pooled). The hepatoma incidence in the control, low-dose drinking water, high-dose drinking water, and test-diet group was 3/24

(12.5%), 3/28 (10.7%), 3/22 (13.6%), and 7/23 (30.4%), respectively.

Incidence of malignant lymphoma in the control, low-dose drinking water, high-dose drinking water, and test-diet groups was 3/24 (12.5%), 4/28 (14.3%), 2/22 (9%), and 2/23 (8.7%), respectively. Incidence of lung adenoma in the

control, low-dose drinking water, high-dose drinking water, and test-diet groups was 10/24 (41.7%), 9/28 (32.1%), 5/22 (22.7%), and 9/23 (39.1%),

respectively. None of these were significantly elevated in a statistical

analysis of this data performed by the EPA. In a 14-month study conducted

with 150 C3H mice (sex not reported), administration of 500 mg/L zinc sulfate

(approximately 100 mg/kg/day) in the drinking water resulted in hypertrophy of

the adrenal cortex and pancreatic islets (Aughey et al., 1977). No tumors

were noted; however, only the adrenal, pancreas and adenohypophysis were

examined. Accurate consumption data could not be obtained due to spillage

during drinking. No instances of adrenal or pancreatic hypertrophy were seen

in a control group (number of animals not stated) that received only distilled



water.

After an intratesticular injection of zinc, Guthrie observed seasonally-related testicular tumors in fowl (Guthrie, 1964) but no tumors in rats

(Guthrie, 1956). Guthrie (1964) administered zinc chloride, zinc acetate or zinc stearate to groups of white leghorn chickens by intratesticular injection

(approximately 0.01 g/injection); groups of chickens were sacrificed from 3

weeks to 11 months. Eight of the 111 chickens injected with zinc chloride in

January and February developed testicular testoma, while none of the 48

chickens injected with zinc chloride in March developed tumors. None of the

36 chickens injected with zinc acetate in March and none of the 14 chickens

injected with zinc stearate in January and February developed tumors; no

conclusions about the carcinogenicity of these two compounds could be made

because an insufficient number of chickens were tested. No control group was

described.

Guthrie injected 0.15-0.20 mL of 10% zinc sulfate into the testis of

nineteen 4-month-old rats and 0.15 mL of 5% zinc chloride into the testis of

twenty-nine 3-month-old rats (strain not specified) (Guthrie 1956). No

testicular tumors were observed in either group at sacrifice 15 months after

injection. No controls were described. Riviere et al. (1959) injected 5%

zinc chloride in distilled water into the testicles of 100 Wistar rats. The

rats were subdivided into several groups; some rats were unilaterally

castrated and some rats received an injection of 200 units serum gonadotrophin

and a subcutaneous implantation of a 25 mg pellet of distilbene or 100 mg

testosterone. The number of rats in each of the four groups (unilateral

castration +/- hormone treatment and untreated +/- hormone treatment was not

stated. No control group was described. Testicular tumors (including

interstitial tumors, a seminoma and an embryoma) became apparent 15 months

after inoculation (tumor incidence not specified). There are no specific data on the effects of hormones in this experiment.

Halme (1961) exposed tumor-resistant and tumor-susceptible strains of mice to zinc in drinking water. In a 3-year, five-generation study zinc chloride was added to the water of tumor-resistant mice (strain not specified); the groups received 0, 10, 20, 50, 100, or 200 mg Zn/L. The spontaneous tumor

frequency for this strain of mice was 0.0004%. The tumor frequencies in the generations were: F0=0.8%, F1=3.5%, F1 and F2=7.6% and F3 and F4=25.7%. Most of the tumors occurred in the 10 and 20 mg Zn dose groups. No statistical analyses and no individual tumor-type data were reported. In the tumor-susceptible mice, strains C3H and A/Sn received 10-29 mg Zn/L in their drinking water for 2 years; 33/76 tumors were observed in the C3H strain (31 in females) and 24/74 tumors were observed in the A/Sn strain (20 in females). Most of the tumors were adenocarcinomas. The numbers of specific tumor types were not reported. The tumor frequencies (43.4% for C3H and 32.4% for A/Sn both sexes combined) were higher than the spontaneous frequency (15% for each strain), although no statistical analyses were reported.

#### II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

In a short-term, in vivo assay, Stoner et al. (1976) injected strain A/Strong mice (20/sex/dose) intraperitoneally with zinc acetate 3 times/week for a total of 24 injections (total doses were 72, 180, or 360 mg/kg). Controls (20/sex/group) consisted of an untreated group, a vehicle control group administered 24 injections of saline and a positive control group administered a single injection of urethan (20 mg/mouse). Mice were sacrificed 30 weeks after the first injection; survival was comparable for all groups. There was no increase in number of lung tumors per mouse in treated animals relative to the pooled controls. While four thymomas

were observed in

zinc acetate-treated groups and none in controls, the occurrence of these

tumors was not statistically significantly elevated.

Urine samples from subjects occupationally exposed in the rubber industry

to a variety of compounds, including zinc oxide, were not found to be

mutagenic in the microtitre fluctuation assay with *Salmonella typhimurium*

strains TA1535, TA98 and TA100 (Crebelli et al., 1985).

The results of short-term genotoxicity assays for zinc are equivocal.

Zinc acetate and/or zinc 2,4-pentanedione have been analyzed in four short-

term mutagenicity assays (Thompson et al., 1989). In the *Salmonella* assay

(with or without hepatic homogenates), zinc acetate was not mutagenic over a

dose range of 50-7200 ug/plate but zinc 2,4-pentanedione was mutagenic to

strains TA1538 and TA98 at 400 ug/plate. The addition of hepatic homogenates

diminished this response in a dose-dependent manner. In the mouse lymphoma

assay, zinc acetate gave a dose-dependent positive response with or without

metabolic activation; the mutation frequency doubled at 10 ug/mL. In the CHO

in vitro cytogenetic assay, zinc acetate gave a dose-dependent positive

response with or without metabolic activation, but the presence of hepatic

homogenates decreased the clastogenic effect. Neither zinc acetate nor zinc

2,4-pentanedione were positive in the unscheduled DNA synthesis assay in rat

hepatocytes over a dose range of 10-1000 ug/mL.

Zinc chloride is reported to be positive in the *Salmonella* assay (Kalinina

et al., 1977), negative in the mouse lymphoma assay (Amacher and Paillet,

1980), and a weak clastogen in cultured human lymphocytes (Deknuddt and

Deminatti, 1978). Zinc sulfate is reported to be not mutagenic in the

*Salmonella* assay (Gocke et al., 1981), and zinc acetate is reported to not

induce chromosomal aberrations in cultured human lymphocytes (Gasiorek and

Bauchinger, 1981). Crebelli et al. (1985) found zinc oxide (99%

purity)  
(1000-5000 ug/plate) to be not mutagenic for Salmonella in the  
reversion  
assay.

Responses in mutagenicity assays are thought to depend on  
the form (e.g.,  
inorganic or organic salt) of the zinc tested. For example,  
inorganic salts  
tend to dissociate and the zinc becomes bound with culture media  
constituents.  
Salts that dissociate less readily tend to be transported into  
the cell and  
are postulated to cause a positive response (Thompson et al.,  
1989). Zinc is  
an essential trace element involved in numerous biological  
functions including  
growth, taste and spermatogenesis. It is a cofactor for several  
enzymes such  
as those involved in the metabolism of proteins and nucleic  
acids. Zinc may  
be a modifier of the carcinogenic response; zinc deficiency or  
excessively  
high levels of zinc may enhance susceptibility to  
carcinogenesis, whereas  
supplementation with low to moderate levels of zinc may offer  
protection (Woo  
et al., 1988). Zinc deficiency enhanced carcinomas of the  
esophagus induced  
by methylbenzyl nitrosoamine (Fong et al., 1978) but retarded the  
development  
of cancer of the oral cavity induced by 4-nitroquinoline-N-oxide  
(Wallenius et  
al., 1979). In a study that examined both zinc deficiency and  
supplementation, Mathur (1979) found that animals with a  
deficient diet (5.9  
mg/kg) and animals diet supplemented with excessively high  
levels of zinc in  
the diet (200-260 mg/kg) had fully developed carcinomas of the  
palatal  
mucosa. While the rats were on the specific diets, the palatal  
mucosa was  
painted with 4 nitroquinoline 3 times/week for 20 weeks. In the  
zinc  
deficient group 2/25 rats developed cancer of the palatal  
mucosa; 2/25 rats  
in the excessive zinc group also developed this form of cancer.  
Animals  
supplemented with moderate levels of zinc in the diet (50 mg/kg)  
developed  
only moderate dysplasia. Thus, zinc's modifying effect on  
carcinogenesis may  
be dose-dependent.

**II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE**

None.

**II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE**

None.

**II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)**

**II.D.1. EPA DOCUMENTATION**

U.S. EPA. 1980. Ambient Water Quality Criteria for Zinc. Prepared by the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-079.

U.S. EPA. 1984. Health Effects Assessment for Zinc (and Compounds). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Emergency and Remedial Response, Washington, DC.

U.S. EPA. 1987. Summary Review of the Health Effects Associated with Zinc and Zinc Oxide. Health Issue Assessment. Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA/600/8-87/022F.

U.S. EPA. 1988. Ambient Water Quality Criteria Document Addendum for Zinc. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards, Washington, DC.

**II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)**

The 1984 Health Effects Assessment for Zinc (and compounds), the 1987 Health Issue Assessment and the 1980 and 1988 Ambient Water

**Quality Criteria**

Documents have received Office of Health Effects Assessment review.

Agency Work Group Review: 11/08/89, 06/15/90

Verification Date: 06/15/90

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Rita S. Schoeny / ORD -- (513)569-7544 / FTS 684-7544

**APPENDIX O**  
**HUMAN HEALTH RISK CALCULATIONS**

**Table O-1**  
**Compounds Detected**  
**Propellant Burning Ground Surface Soil (0-2')**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u> <u>(Y/N)?</u>	<u>Reason *</u>	<u>Exposure Point</u> <u>Concentration **</u>
24DNT	16 : 114	53.3	2.77	Y		10.7
26DNT	2 : 114	4.25	3.41	Y		1
2MNAP	2 : 13	0.452	0.122	Y		0.452
ACET	1 : 58	0.006	-	N	4	
AG	3 : 108	25.8	2.02	N	4	
AS	83 : 108	64	2.88	Y		9.45
B2EHP	1 : 13	6.2	-	Y		6.2
BAANTR	1 : 13	0.204	-	Y		0.204
BE	81 : 108	2.29	0.494	N	1	
C6H6	8 : 114	2.64	0.199	Y		0.42
CCL3F	4 : 114	0.005	0.003	N	4	
CD	3 : 108	4.48	1.7	N	4	
CHRY	1 : 13	3.68	-	Y		3.68
CR	108 : 108	89.8	7.15	Y		49.8
CU	108 : 108	2700	9.57	Y		344
DEP	7 : 13	6.2	0.568	Y		6.2
DNBP	4 : 13	6.35	2.06	Y		6.35
FANT	2 : 13	0.2	0.145	Y		0.2
HG	31 : 108	7.7	0.058	Y		0.334
MEK	7 : 64	0.01	0.006	N	2	
NI	108 : 108	63.9	6.57	Y		27.3
NNDPA	3 : 13	30.8	1.22	Y		30.8
PB	108 : 108	3300	12	Y		2700
PHANTR	3 : 13	1.32	0.11	Y		1.32
PYR	1 : 13	0.168	-	Y		0.168
SB	1 : 108	404	-	N	4	
SE	10 : 108	2.03	0.581	Y		0.618
TL	2 : 108	2.28	1.19	N	1, 4	
ZN	108 : 108	5200	27	Y		1040

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant.  
                   \* 3 = essential for human nutrition.  
                   \* 4 = frequency of detection less than 5 %.  
                   \*\* 95th percentile or maximum

Note:           Assessment of surface soil contamination was performed using samples PBS-91-01 through PBS-91-108. In addition, the upper portions of samples PBS-91-109 through PBS-91-114 were used to assess contamination of surface soil by 24DNT, 26DNT, C6H6, and CCL3F.



Table O-2  
Compounds Detected  
Propellant Burning Ground Subsurface Soil (0 - 12')  
Units: ug/g

Remedial Investigation  
Badger Army Ammunition Plant

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment (Y/N)?</u>	<u>Reason *</u>	<u>Exposure Point Concentration **</u>
111TCE	1 : 24	0.002	-	N	4	
12DCE	1 : 24	10	-	N	4	
24DNT	6 : 45	73000	6.127	Y		58.9
26DNT	2 : 45	6.2	5.96	Y		2
2MNAP	2 : 7	18.2	1.65	Y		18.2
4E2MHX	1 : 24	8.36	-	N	4	
ACET	1 : 23	0.002	-	N	4	
AG	2 : 30	1.05	0.976	N	1	
AL	2 : 2	3234.706	2082.747	N	1	
ANAPNE	1 : 7	16.9	-	Y		16.9
ANAPYL	1 : 7	1.04	-	Y		1.04
ANTRC	1 : 7	12.4	-	Y		12.4
AS	17 : 30	29.5	3.1	Y		18.8
B2EHP	1 : 7	6.2	-	Y		6.2
BA	2 : 2	16.532	12.644	N	1	
BAANTR	1 : 7	8.9	-	Y		8.9
BAPYR	1 : 7	3.55	-	Y		3.55
BBFANT	1 : 7	3.91	-	Y		3.91
BE	18 : 30	1.46	0.497	N	1	
BGHIPI	1 : 7	2.57	-	Y		2.57
BKFANT	1 : 7	3.36	-	Y		3.36
C6H6	5 : 24	864	0.001	Y		9.09
CA	2 : 2	104249.93	40785.414	N	3	
CCL4	2 : 46	0.645	0.39	N	4	
CD	1 : 30	1.66	-	N	4	
CH2CL2	1 : 24	0.825	-	N	4	
CHRY	1 : 7	8.28	-	Y		8.28
CO	2 : 2	4.199	3.752	N	1	
CR	30 : 30	63.3	4.31	Y		40.4
CU	51 : 52	5945.474	4.161	Y		327.19
DBAHA	1 : 7	0.661	-	Y		0.661
DBZFUR	1 : 7	5.8	-	Y		5.8
DEP	2 : 7	6.2	-	Y		6.2
DNBP	1 : 7	6.2	-	Y		6.2
ETC6H5	1 : 24	1.54	-	N	4	
FANT	1 : 7	6.2	-	Y		6.2
FE	2 : 2	11556.239	7435.939	N	1,3	
FLRENE	1 : 7	18.4	-	Y		18.4
HG	4 : 30	0.16	0.06	N	1	
ICDPYR	1 : 7	4.52	-	Y		4.52
K	2 : 2	510.007	285.424	N	1,3	

continued

**Table O-2**  
**Compounds Detected**  
**Propellant Burning Ground Subsurface Soil (0 - 12')**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment (Y/N)?</u>	<u>Reason *</u>	<u>Exposure Point Concentration **</u>
MEC6H5	3 : 24	14.4	1.04	Y		14.4
MEK	5 : 23	2.67	0.003	N	2	
MG	2 : 2	59777.063	22398.287	N	3	
MIBK	1 : 23	4.99	-	N	4	
MN	2 : 2	280.985	216.649	N	1	
NA	2 : 2	379.441	222.867	N	1,3	
NAP	1 : 7	6.2	-	Y		6.2
NI	30 : 30	27.9	4.27	N	1	
NIT	18 : 18	35	1.16	Y		35
NNDPA	1 : 7	12	-	Y		12
PB	46 : 52	5371.52	1.55	Y		1200
PHANTR	1 : 7	12	-	Y		12
PYR	1 : 7	6.2	-	Y		6.2
SE	3 : 30	1.77	0.585	Y		1.77
SO4	13 : 18	280	8.55	Y		280
TCLEE	2 : 46	1.14	0.334	N	4	
TRCLE	4 : 46	39.4	0.003	Y		0.23
TXYLEN	2 : 8	39.5	11.5	Y		39.5
V	2 : 2	27.618	15.014	N	1	
ZN	52 : 52	2984.426	3.95	Y		1253.94

Footnotes: \* 1 = within background range.  
 \* 2 = laboratory or sampling contaminant.  
 \* 3 = essential for human nutrition.  
 \* 4 = frequency of detection less than 5 %.  
 \*\* 95th percentile or maximum

Note: Assessment of subsurface soil contamination from 0 to 12 feet was performed using data from the following borings, test pits, and surface soil samples:  
 LOB-90-01, LOB-90-02, PBB-90-01, PBB-90-02, PBT-90-01 through PBT-90-08, PBB-91-01 through PBB-91-07, and the deeper samples from PBS-91-109 through PBS-91-118.

TABLE O-3

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
PROPELLANT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

P8083308

25-Mar-93

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS		mg/kg	Calculator
INGESTION RATE - ADULT	IRa	100	mg/day	USEPA, 1991
INGESTION RATE - CHILD	IRc	200	mg/day	USEPA, 1991
FRACTION INGESTED	FI	100%		Assumption
CONVERSION FACTOR	CF	0.000001	kg/mg	USEPA, 1991
BODY WEIGHT - ADULT	BW <sub>a</sub>	70	kg	USEPA, 1991
BODY WEIGHT - CHILD	BW <sub>c</sub>	15	kg	USEPA, 1991
EXPOSURE FREQUENCY	EF	350	days/year	USEPA, 1991
EXPOSURE DURATION - ADULT	ED <sub>a</sub>	24	years	USEPA, 1991
EXPOSURE DURATION - CHILD	ED <sub>c</sub>	6	years	USEPA, 1991
AVERAGING TIME				
CANCER	AT	70	years	USEPA, 1991
ADULT - NONCANCER	AT <sub>a</sub>	24	years	USEPA, 1991
CHILD - NONCANCER	AT <sub>c</sub>	6	years	USEPA, 1991
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	USEPA, 1991
Note: For noncarcinogenic effects: AT = ED				
CANCER RISK = INTAKE (mg/kg-day) x CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup> HAZARD QUOTIENT = INTAKE (mg/kg-day) / REFERENCE DOSE (mg/kg-day) INTAKE-ADULT = $\frac{CS \times IR_a \times FI \times CF \times EF \times ED_a}{BW_a \times AT_a \times 365 \text{ days/yr}}$ INTAKE-CHILD = $\frac{CS \times IR_c \times FI \times CF \times EF \times ED_c}{BW_c \times AT_c \times 365 \text{ days/yr}}$				

USEPA, 1991. Risk Assessment Guidelines for Superfund  
USEPA, 1991. Standard Default Exposure Factors

TABLE O-3, continued

INCIDENTAL INGESTION OF SURFACE SOIL.  
RESIDENTIAL - ADULT AND CHILD  
PROPELLANT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION BAP	INTAKE ADULT (mg/kg-day)	INTAKE CHILD (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK ADULT	CANCER RISK CHILD	TOTAL CANCER RISK
2,4-DNT	10.7	1	5.0E-06	1.2E-03	6.8E-01	3.4E-06	8.0E-06	1.1E-05
2,6-DNT	1	1	4.7E-07	1.1E-06	6.8E-01	3.2E-07	7.3E-07	1.1E-06
AS	9.45	1	4.4E-06	1.0E-03	1.8E+00	8.0E-06	1.9E-05	2.7E-05
BZ	6.2	1	2.9E-06	6.8E-06	1.4E-02	4.1E-08	9.5E-08	1.4E-07
BAA/NTA	0.204	1	9.6E-08	2.2E-07	7.3E+00	7.0E-07	1.6E-06	2.3E-06
CB	0.42	1	2.0E-07	4.8E-07	2.9E-02	5.7E-09	1.3E-08	1.9E-08
CHRY	3.68	1	1.7E-06	4.0E-06	7.3E+00	1.3E-05	2.9E-05	4.2E-05
CR	49.8	1	2.3E-05	5.5E-03	ND			
INDPA	30.8	1	1.4E-05	3.4E-03	4.9E-03	7.1E-08	1.7E-07	2.4E-07
PS	2700	1	1.5E-03	3.0E-03	ND			
SUMMARY CANCER RISK						3E-05	6E-05	8E-05

TABLE O-3, continued

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
PROPELLANT BURNING GROUND  
BADOER ARMY AMMUNITION PLANT

## NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION BAF	INTAKE ADULT (mg/kg-day)	INTAKE CHILD (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT ADULT	HAZARD QUOTIENT CHILD	TOTAL HAZARD QUOTIENT
ADMT	10.7	1	1.5E-03	1.4E-04	2.0E-03	7.33E-03	6.84E-02	7.57E-02
ADMT	1	1	1.4E-06	1.3E-05	ND			
ADMP	0.452	1	6.2E-07	5.8E-06	4.0E-02	1.53E-05	1.44E-04	1.60E-04
AS	9.45	1	1.3E-05	1.2E-04	3.0E-04	4.32E-02	4.03E-01	4.46E-01
BZEP	6.2	1	8.5E-06	7.9E-05	2.0E-02	4.25E-04	3.90E-03	4.39E-03
BAANTH	0.204	1	2.8E-07	2.6E-06	4.0E-02	6.99E-06	6.52E-05	7.22E-05
CBM	0.42	1	5.8E-07	5.4E-06	ND			
CHRY	3.68	1	5.0E-06	4.7E-05	4.0E-02	1.20E-04	1.18E-03	1.30E-03
CR	49.8	1	6.8E-05	6.4E-04	5.0E-03	1.36E-02	1.27E-01	1.41E-01
CU	344	1	4.7E-04	4.4E-03	ND			
DEP	6.2	1	8.5E-06	7.9E-05	8.0E-01	1.06E-05	9.91E-05	1.10E-04
DNEP	6.55	1	8.7E-06	8.1E-05	1.0E-01	8.70E-05	8.12E-04	8.99E-04
PANT	0.2	1	2.7E-07	2.6E-06	4.0E-02	6.85E-06	6.39E-05	7.08E-05
BO	0.354	1	4.6E-07	4.3E-06	3.0E-04	1.53E-03	1.42E-02	1.58E-02
NI	27.3	1	3.7E-05	3.5E-04	2.0E-02	1.87E-03	1.73E-02	1.93E-02
INDPA	30.8	1	4.2E-05	3.9E-04	ND			
PS	2700	1	3.7E-03	3.5E-02	ND			
PLANTH	1.52	1	1.8E-06	1.7E-05	4.0E-02	4.52E-05	4.22E-04	4.67E-04
PTK	0.168	1	2.3E-07	2.1E-06	3.0E-02	7.87E-06	7.16E-05	7.93E-05
SE	0.618	1	8.5E-07	7.9E-06	5.0E-03	1.69E-04	1.58E-03	1.75E-03
ZN	1040	1	1.4E-03	1.3E-02	2.0E-01	7.12E-03	6.63E-02	7.34E-02
SUMMARY HAZARD INDEX								
						0.8735	0.7851	0.7406

TABLE O-4

## INCIDENTAL INGESTION AND INHALATION OF SURFACE SOIL

GROUNDS MAINTENANCE WORKER  
 PROPELLANT BURNING GROUND  
 BADGER ARMY AMMUNITION PLANT

PROSSCM      06-Dec-92

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	95th Percentile	mg/kg	USEPA, 1991a
INGESTION RATE	IR	100	mg/day	Assumption
FRACTION INGESTED	FI	100%		
CONVERSION FACTOR	CF	0.000001	kg/mg	
BODY WEIGHT	BW	70	kg	USEPA, 1991a
EXPOSURE FREQUENCY	EF	24	days/year	USEPA, 1991a
EXPOSURE DURATION	ED	25	years	USEPA, 1991a
CONCENTRATION AIR PARTICULATES	CAp	Calculated	mg/m <sup>3</sup>	Appendix M
CONCENTRATION AIR VOLATILES	CAV	Calculated	mg/m <sup>3</sup>	USEPA, 1991b
VOLATILIZATION FACTOR	VF	Calculated	m <sup>3</sup> /kg	USEPA, 1991a
PARTICULATE EMISSION FACTOR	PEF	4.63E+09	m <sup>3</sup> /hour	Assumption
INHALATION RATE	IR	2.5	m <sup>3</sup> /hour	
EXPOSURE TIME	ET	8	hours/day	
AVERAGING TIME				
RELATIVE ABSORPTION FACTOR				
CANCER	AT	70	years	USEPA, 1989
NONCANCER	AT	25	years	USEPA, 1991a
	RAF	1	unitless	USEPA, 1989

USEPA, 1989 Risk Assessment Guidelines for Superfund-Part A

USEPA, 1990 Exposure Factors Handbook

USEPA, 1991a Standard Default Exposure Factors

USEPA, 1991b Risk Assessment Guidelines for Superfund-Part B

Note:

For noncarcinogenic effects: AT = ED

$$\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$$

$$\text{HAZARD QUOTIENT}_{\text{Ingestion}} = \text{INTAKE (mg/kg-day)} / \text{REFERENCE DOSE (mg/kg-day)}$$

$$\text{HAZARD QUOTIENT}_{\text{Inhalation}} = \frac{\text{AIR CONCENTRATION (mg/m}^3\text{)}}{\text{REFERENCE CONCENTRATION (mg/m}^3\text{)}}$$

$$\text{INTAKE-INGESTION} = \frac{\text{CS} \times \text{IR} \times \text{RAF} \times \text{FI} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

$$\text{INTAKE-INHALATION} = \frac{(\text{CAp} \times \text{CAV}) \times \text{IR} \times \text{ET} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

$$\text{AIR CONCENTRATION (mg/m}^3\text{)} = \text{CAp} \div \text{CAF}$$

$$\text{CAp} = \text{CS} \times \text{VF} \times \text{RAF}$$

$$\text{CAF} = \text{CS} \times \text{VF}$$

TABLE O-4, continued

INCIDENTAL INGESTION AND INHALATION OF SURFACE SOIL,  
 GROUNDS MAINTENANCE WORKER  
 PROPELLANT BURNING GROUND  
 BADGER ARMY AMMUNITION PLANT

FBGSSOM 06-Dec-92

## CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION EAP	INTAKE		CANCER SLOPE FACTOR - INH. (mg/kg-dm) <sup>-1</sup>	CANCER SLOPE FACTOR - ING. (mg/kg-dm) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK INHALATION	TOTAL CANCER RISK
			INGESTION (mg/kg-dm)	INHALATION (mg/kg-dm)					
2,4-DNT	10.7	1	3.4E-07	1.6E-11	ND	6.8E-01	2.4E-07		2.4E-07
2,4-DNT	1	1	3.4E-06	1.4E-12	ND	6.8E-01	2.3E-08		2.3E-08
AS	9.45	1	3.2E-07	1.4E-11	5.0E+01	1.8E+00	5.7E-07	6.8E-10	5.7E-07
BZEHIP	6.2	1	2.1E-07	9.0E-12	ND	1.4E-02	2.9E-09		2.9E-09
BAA/NTR	0.204	1	6.8E-09	3.0E-13	6.1E+00	7.3E+00	5.0E-08	1.8E-12	5.0E-08
CHH	0.42	1	1.4E-08	6.7E-07	2.9E-02	2.9E-02	4.1E-10	1.9E-08	2.0E-08
CHRY	3.68	1	1.2E-07	5.3E-12	6.1E+00	7.3E+00	9.0E-07	3.3E-11	9.0E-07
CR	49.8	1	1.7E-06	7.2E-11	4.1E+01	ND	5.1E-09	3.0E-09	5.1E-09
MNDPA	30.8	1	1.0E-06	4.5E-11	ND	4.9E-03			
PB	2700	1	9.1E-05	3.9E-09	ND	ND			
SUMMARY CANCER RISK									2E-06

TABLE O-4, continued

INCIDENTAL INGESTION AND INHALATION OF SURFACE SOIL,  
 GROUNDS MAINTENANCE WORKER  
 PROPELLANT BURNING GROUND  
 BADGER ARMY AMMUNITION PLANT

NONCARCINOGENIC EFFECTS

PROSSGM 06-Dec-92

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION BAF	INTAKE INGESTION (mg/kg-day)	AIR CONCENTRATION (mg/m <sup>3</sup> )	REFERENCE CONC. (mg/m <sup>3</sup> )	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT INHALATION	TOTAL HAZARD QUOTIENT
24DNT	10.7	1	1.0E-06	2.5E-09	ND	2.0E-03	5.03E-04		5.03E-04
26DNT	1	1	9.4E-08	2.2E-10	ND	ND			
2MNAP	0.452	1	4.2E-08	9.8E-11	ND	4.0E-02	1.06E-06		1.06E-06
AS	9.45	1	8.9E-07	2.0E-09	ND	3.0E-04	2.96E-03		2.96E-03
B2EHP	6.2	1	5.8E-07	1.3E-09	ND	2.0E-02	2.91E-05		2.91E-05
BAANTR	0.204	1	1.9E-08	4.4E-11	ND	4.0E-02	4.79E-07		4.79E-07
C6H6	0.42	1	3.9E-08	1.0E-04	ND	ND			
CHRY	3.68	1	3.5E-07	7.9E-10	ND	4.0E-02	8.64E-06		8.64E-06
CR	49.8	1	4.7E-06	1.1E-08	ND	5.0E-03	9.36E-04		9.36E-04
CU	344	1	3.2E-05	7.4E-08	ND	ND			
DEP	6.2	1	5.8E-07	1.3E-09	ND	8.0E-01	7.28E-07		7.28E-07
DNBP	6.35	1	6.0E-07	1.4E-09	ND	1.0E-01	5.96E-06		5.96E-06
FANT	0.2	1	1.9E-08	4.3E-11	ND	4.0E-02	4.70E-07		4.70E-07
HQ	0.334	1	3.1E-08	7.2E-11	3.0E-04	3.0E-04	1.05E-04	2.40E-07	1.05E-04
NI	27.3	1	2.6E-06	5.9E-09	ND	2.0E-02	1.28E-04		1.28E-04
NNDPA	30.8	1	2.9E-06	6.7E-09	ND	ND			
PB	2700	1	2.5E-04	5.8E-07	ND	ND			
PHANTR	1.32	1	1.2E-07	2.9E-10	ND	4.0E-02	3.10E-06		3.10E-06
PYR	0.168	1	1.6E-08	3.6E-11	ND	3.0E-02	5.26E-07		5.26E-07
SE	0.618	1	5.8E-08	1.3E-10	ND	5.0E-03	1.16E-05		1.16E-05
ZN	1040	1	9.8E-05	2.2E-07	ND	2.0E-01	4.88E-04		4.88E-04
SUMMARY HAZARD INDEX							0.0052	0.0000	0.0052



TABLE O-3

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SURFACE SOIL  
FARMER  
PROPELLANT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

PROCESS-P 08-Dec-92

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	95th Percentile	mg/kg	Calculator
INGESTION RATE	IR	480	mg/day	USEPA, 1991
FRACTION INGESTED	FI	100%		Assumptions
SOIL ADHERENCE FACTOR	SAF	1	mg/cm <sup>2</sup>	USEPA, 1992
SURFACE AREA EXPOSED	SA	2,100	cm <sup>2</sup> /day	USEPA, 1990
CONVERSION FACTOR	CF	0.000001	kg/mg	
BODY WEIGHT	BW	70	kg	USEPA, 1991
EXPOSURE FREQUENCY	EF	24	days/year	USEPA, 1991
EXPOSURE DURATION	ED	30	years	USEPA, 1991
AVERAGING TIME				
CANCER	AT	70	years	USEPA, 1989
NONCANCER	AT	30	years	USEPA, 1991
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	USEPA, 1989
INGESTION				
DERMAL				
USEPA, 1989. Risk Assessment Guidance for Superfund				
USEPA, 1990. Exposure Factors Handbook				
USEPA, 1991. Standard Default Exposure Factors				
USEPA, 1992. Dermal Exposure Guidance				

Note:

For noncarcinogenic effects: AT = ED

CANCER RISK = INTAKE (mg/kg-day) × CANCER SLOPE FACTOR (mg/kg-day)<sup>-1</sup>

HAZARD QUOTIENT = INTAKE (mg/kg-day) / REFERENCE DOSE (mg/kg-day)

INTAKE = (INTAKE-INGESTION) + (INTAKE-DERMAL)

INTAKE-INGESTION =  $\frac{CS \times IR \times FI \times CF \times EF \times ED}{BW \times AT}$

INTAKE-DERMAL =  $\frac{CS \times SAF \times RA \times CF \times EF \times ED}{BW \times AT}$

TABLE O-5, continued

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SURFACE SOIL  
FARMER  
PROPELLANT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

## CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RPF	INTAKE INGESTION (mg/kg-day)	DERMAL RPF	INTAKE DERMAL (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK DERMAL	TOTAL CANCER RISK
24DNT	10.7	1	2.1E-06	No values available for Quantitative Analysis		6.8E-01	1.4E-06		1.4E-06
26DNT	1	1	1.9E-07			6.8E-01	1.3E-07		1.3E-07
AS	9.45	1	1.8E-06			1.8E+00	3.3E-06		3.3E-06
B2EHF	6.2	1	1.2E-06			1.4E-02	1.7E-08		1.7E-08
BAANTF	0.204	1	3.9E-06			7.3E+00	2.9E-07		2.9E-07
CB16	0.42	1	8.1E-06			2.9E-02	2.4E-09		2.4E-09
CHRY	3.68	1	7.1E-07			7.3E+00	5.2E-06		5.2E-06
CR	49.8	1	9.8E-06			ND	2.9E-06		2.9E-06
NNDPA	30.8	1	6.0E-06			4.9E-03			
PB	2700	1	5.2E-04			ND			
SUMMARY CANCER RISK							1E-05	9E+00	1E-05

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SURFACE SOIL  
FARMER  
PROPELLANT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAP	INTAKE INGESTION (mg/kg-day)	DERMAL RAP	INTAKE DERMAL (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT DERMAL	TOTAL HAZARD QUOTIENT
24DNT	10.7	1	4.8E-06	No values available for Quantitative Analysis		2.0E-03	2.41E-03		2.41E-03
26DNT	1	1	4.5E-07			ND			
2MNAP	0.452	1	2.0E-07			4.0E-02	5.09E-06		5.09E-06
AS	9.45	1	4.3E-06			3.0E-04	1.42E-02		1.42E-02
B2HP	6.2	1	2.8E-06			2.0E-02	1.40E-04		1.40E-04
BAANTR	0.204	1	9.2E-08			4.0E-02	2.30E-06		2.30E-06
C6H6	0.42	1	1.9E-07			ND			
CHRY	3.68	1	1.7E-06			4.0E-02	4.15E-05		4.15E-05
CR	49.8	1	2.2E-05			5.0E-03	4.49E-03		4.49E-03
CU	344	1	1.6E-04			ND			
DEP	6.2	1	2.8E-06			8.0E-01	3.49E-06		3.49E-06
DNBP	6.35	1	2.9E-06			1.0E-01	2.86E-05		2.86E-05
FANT	0.2	1	9.0E-08			4.0E-02	2.25E-06		2.25E-06
HO	0.334	1	1.5E-07			3.0E-04	5.02E-04		5.02E-04
NI	27.3	1	1.2E-05			2.0E-02	6.15E-04		6.15E-04
NNDPA	30.8	1	1.4E-05			ND			
PB	2700	1	1.2E-03			ND			
PHANTR	1.32	1	6.0E-07			4.0E-02	1.49E-05		1.49E-05
PYR	0.168	1	7.6E-08			3.0E-02	2.52E-06		2.52E-06
SE	0.618	1	2.8E-07			5.0E-03	5.57E-05		5.57E-05
ZN	1040	1	4.7E-04			2.0E-01	2.34E-03		2.34E-03
SUMMARY HAZARD INDEX							0.0249	0.0000	0.0249

TABLE O-6

INITIALATION EXPOSURE TO AMBIENT AIR  
FARMER  
PROPELLANT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

FRABP 08-Dec-92

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	95th Percentile Calculated	mg/kg	see below
CONCENTRATION AIR PARTICULATES	CAP	Calculated	mg/m <sup>3</sup>	see below
CONCENTRATION AIR VOLATILES	CAV	Calculated	mg/m <sup>3</sup>	Appendix M
VOLATILIZATION FACTOR	VF	Calculated	m <sup>3</sup> /kg	Appendix M
PM10 DURING TILLING	PM10	8100	ug/m <sup>3</sup>	
CONVERSION FACTOR	CF	1E-09	kg/kg	
INHALATION RATE	IR	2.5	m <sup>3</sup> /hour	USEPA, 1991
BODY WEIGHT	BW	70	kg	USEPA, 1989
EXPOSURE TIME	ET	8	hour/day	Assumption
EXPOSURE FREQUENCY	EF	10	day/year	Assumption
EXPOSURE DURATION	ED	30	years	Assumption
AVAILABILITY TIME	AT	70	years	USEPA, 1991
CANCER	AI	30	years	USEPA, 1991
NONCANCER				

USEPA, 1989. Risk Assessment Guidance for Superfund, Part A

USEPA, 1991. Standard Default Exposure Factors

$$\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$$

$$\text{HAZARD QUOTIENT} = \text{CAP OR CAV (mg/m}^3\text{)} / \text{REFERENCE CONCENTRATION (mg/m}^3\text{)}$$

$$\text{INTAKE} = \frac{(\text{CAP} \times \text{CAV}) \times \text{IR} \times \text{ET} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ day/yr}}$$

$$\text{AIR CONCENTRATION PARTICULATES} = \text{CS} \times \text{PM10} \times \text{CF}$$

$$\text{AIR CONCENTRATION VOLATILES} = \text{CS} \times \text{VF}$$

TABLE O-4, continued  
 INITIALATION EXPOSURE TO AMBIENT AIR  
 FARMER  
 PROPELLANT BURNING GROUND  
 BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VP (m³/kg)	AIR CONCENTRATION VOLATILES (mg/m³)	AIR CONCENTRATION PARTICULATES (mg/m³)	INTAKE (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day)⁻¹	CANCER RISK
2,4-DNT	10.7			0.0000667	2.9E-07	ND	
2,6-DNT	1			0.0000081	2.7E-08	ND	
AS	9.43			0.00076545	2.6E-07	5.0E+01	1.3E-05
BZEMP	6.2			0.00035022	1.7E-07	ND	
BAANTR	0.204			0.0000016524	5.5E-09	6.1E+00	3.4E-08
CBM	0.42	4.22E+03	0.000995261	0.00003402	3.5E-07	2.9E-02	1.0E-08
CBRY	5.68			0.000029808	1.0E-07	6.1E+00	6.1E-07
CR	49.8			0.00040338	1.4E-06	4.1E+01	5.5E-05
MDPA	50.8			0.00024948	8.4E-07	ND	
PS	2700			0.02187	7.3E-05	ND	
SUMMARY CANCER RISK							7E-05

TABLE O-6, continued  
INITIALATION EXPOSURE TO AMBIENT AIR  
FARMER  
PROPELLANT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VF ( $\mu\text{g/g}$ )	AIR CONCENTRATION VOLATILES ( $\text{mg}/\text{m}^3$ )	AIR CONCENTRATION PARTICULATES ( $\text{mg}/\text{m}^3$ )	REFERENCE CONCENTRATION ( $\text{mg}/\text{kg}$ )	HAZARD QUOTIENT VOLATILES	HAZARD QUOTIENT PARTICULATES	HAZARD QUOTIENT TOTAL
ACNT	10.7				0.0000667	ND		
ACNT	1				0.0000081	ND		
BARAP	0.452				0.000004612	ND		
AS	9.45				0.000076545	ND		
BZBP	6.2				0.00005022	ND		
BAANTR	0.204				0.0000016574	ND		
CBP6	0.42	4220	0.000095261		0.000003402	ND		
CBRY	3.66				0.000029808	ND		
CR	49.8				0.00040338	ND		
CU	344				0.0027864	ND		
DPP	6.2				0.00005022	ND		
DHP	6.35				0.000051405	ND		
PAINT	0.2				0.00000162	ND		
EG	0.354				0.0000027054	8.5E-05	3.2E-02	3.2E-02
NI	27.5				0.00022113	ND		
NDPA	30.8				0.00024948	ND		
FB	2700				0.02187	ND		
PLANTR	1.52				0.000010892	ND		
PYR	0.168				0.0000013608	ND		
SE	0.618				0.0000050358	ND		
ZN	1040				0.008424	ND		
SUMMARY HAZARD INDEX						0.00	0.03	0.03

TABLE O-7

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0-12 feet)  
CONSTRUCTION WORKER  
PROPELLANT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

FIGSB-CW 08-Dec-92

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Maximum	mg/kg	USEPA, 1991
INGESTION RATE	IR	480	mg/day	Assumption
FRACTION INGESTED	FI	100%		USEPA, 1992
SOIL ADHERENCE FACTOR	SAF	1	mg/cm <sup>2</sup>	USEPA, 1990
SURFACE AREA EXPOSED	SA	2,100	cm <sup>2</sup> /day	
CONVERSION FACTOR	CF	0.000001	kg/mg	
BODY WEIGHT	BW	70	kg	USEPA, 1991
EXPOSURE FREQUENCY	EF	20	days/year	USEPA, 1991
EXPOSURE DURATION	ED	1	years	USEPA, 1991
AVERAGING TIME	AT	70	years	USEPA, 1989
RELATIVE ABSORPTION FACTOR	RAF	0.0547945205	years	USEPA, 1991
INGESTION DERMAL		1	unitless	USEPA, 1989

USEPA, 1989. Risk Assessment Guidelines for Superfund

USEPA, 1990. Exposure Factors Handbook

USEPA, 1991. Standard Default Exposure Factors

USEPA, 1992. Dermal Exposure Guidelines

Note:

For noncarcinogenic effects: AT =

EF

365 days

CANCER RISK =  $\text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$

HAZARD QUOTIENT =  $\text{INTAKE (mg/kg-day)} / \text{REFERENCE DOSE (mg/kg-day)}$

INTAKE =  $(\text{INTAKE-INGESTION}) + (\text{INTAKE-DERMAL})$

INTAKE-INGESTION =  $\frac{\text{CS} \times \text{IR} \times \text{RAF} \times \text{FI} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ day/yr}}$

INTAKE-DERMAL =  $\frac{\text{CS} \times \text{SA} \times \text{SAF} \times \text{RAF} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ day/yr}}$

TABLE 0-7, continued  
DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0-12 feet)  
CONSTRUCTION WORKER  
PROPELLANT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAP	INTAKE (mg/kg-dm)	DERMAL RAP	INTAKE (mg/kg-dm)	CANCER SLOPE FACTOR (mg/kg-dm) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK DERMAL	TOTAL CANCER RISK
24DNT	58.9	1	3.2E-07	No Values Available for Quantitative Analysis		6.8E-01	2.1E-07		2.1E-07
26DNT	2	1	1.1E-08			6.8E-01	7.3E-09		7.3E-09
AS	18.8	1	1.0E-07			1.8E+00	1.8E-07		1.8E-07
B2HP	6.2	1	3.3E-08			1.4E-02	4.7E-10		4.7E-10
BAANTP	8.9	1	4.8E-08			7.3E+00	3.5E-07		3.5E-07
BAPYR	3.55	1	1.9E-08			7.3E+00	1.4E-07		1.4E-07
BIFANT	3.91	1	2.1E-08			7.3E+00	1.5E-07		1.5E-07
BIFANT	3.36	1	1.8E-08			7.3E+00	1.3E-07		1.3E-07
CH6	9.09	1	4.9E-08			2.9E-02	1.4E-09		1.4E-09
CHRY	8.28	1	4.4E-08			7.3E+00	3.2E-07		3.2E-07
CR	40.4	1	2.2E-07			ND			
DBAH	0.661	1	3.5E-09			7.3E+00	2.6E-08		2.6E-08
ICDPYR	4.52	1	2.4E-08			7.3E+00	1.8E-07		1.8E-07
NNDA	12	1	6.4E-08			7.0E+00	4.5E-07		4.5E-07
PB	1200	1	6.4E-06			ND			
TRCLE	0.23	1	1.2E-09			1.1E-02	1.4E-11		1.4E-11
SUMMARY CANCER RISK							2E-06	0E+00	2E-06



TABLE O-7, continued

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0-12 feet)

CONSTRUCTION WORKER

PROPELLANT BURNING GROUND

BADGER ARMY AMMUNITION PLANT

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAV	INTAKE INGESTION (mg/kg-day)	DERMAL RAV	INTAKE DERMAL (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT DERMAL	TOTAL HAZARD QUOTIENT
24DNT	58.9	1	4.0E-04	No Values Available for Quantitative Analysis		2.0E-03	2.02E-01		2.02E-01
26DNT	2	1	1.4E-05			ND			
2NNAP	18.2	1	1.2E-04			4.0E-02	3.12E-03		3.12E-03
ANAPNE	16.9	1	1.2E-04			4.0E-02	2.90E-03		2.90E-03
ANAPLY	1.04	1	7.1E-06			4.0E-02	1.78E-04		1.78E-04
ANTRC	12.4	1	8.5E-05			4.0E-02	2.13E-03		2.13E-03
AS	18.8	1	1.3E-04			3.0E-04	4.30E-01		4.30E-01
B2E1P	6.2	1	4.3E-05			2.0E-02	2.13E-03		2.13E-03
BAANTR	8.9	1	6.1E-05			4.0E-02	1.53E-03		1.53E-03
BAPYR	3.55	1	2.4E-05			4.0E-02	6.09E-04		6.09E-04
BEFANT	3.91	1	2.7E-05			4.0E-02	6.70E-04		6.70E-04
BOBEPY	2.57	1	1.8E-05			4.0E-02	4.41E-04		4.41E-04
BEFANT	3.36	1	2.3E-05			4.0E-02	5.76E-04		5.76E-04
ORH6	9.09	1	6.2E-05			ND			
CHRY	8.28	1	5.7E-05			4.0E-02	1.42E-03		1.42E-03
CR	40.4	1	2.8E-04			5.0E-03	5.54E-02		5.54E-02
CJ	327.19	1	2.2E-03			ND			
DBA1A	0.661	1	4.5E-06			4.0E-02	1.13E-04		1.13E-04
DEZPUR	5.8	1	4.0E-05			ND			
DEP	6.2	1	4.3E-05			8.0E-01	5.31E-05		5.31E-05
DNEP	6.2	1	4.3E-05			1.0E-01	4.25E-04		4.25E-04
PANT	6.2	1	4.3E-05			4.0E-02	1.06E-03		1.06E-03
FLURENE	18.4	1	1.3E-04			4.0E-02	3.15E-03		3.15E-03
KDPTP	4.52	1	3.1E-05			4.0E-02	7.75E-04		7.75E-04
MEOBUS	1.04	1	7.1E-06			0.2	3.57E-05		3.57E-05
NAP	6.2	1	4.3E-05			0.04	1.06E-03		1.06E-03
NTT	35	1	2.4E-04			0.1	2.40E-03		2.40E-03
NNDPA	12	1	8.2E-05			ND			
PB	1200	1	8.2E-03			ND			

TABLE O-7, continued  
DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0-12 feet)  
CONSTRUCTION WORKER  
PROPELLANT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RPF	INTAKE INGESTION (mg/kg-day)	DERMAL RPF	INTAKE DERMAL (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT DERMAL	TOTAL HAZARD QUOTIENT
PIANTH	12	1	8.2E-05			0.04	2.06E-03		2.06E-03
PYR	6.2	1	4.3E-05			0.03	1.42E-03		1.42E-03
SE	1.77	1	1.2E-05			0.005	2.43E-03		2.43E-03
SO4	280	1	1.9E-03			ND			
TRCLE	0.23	1	1.6E-06			ND			
TXLEN	39.5	1	2.7E-04			2	1.35E-04		1.35E-04
ZN	125394	1	8.6E-03			0.2	4.30E-02		4.30E-02
SUMMARY HAZARD INDEX									0.5
									0.5

TABLE O-7A

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0-12 feet)  
CONSTRUCTION WORKER  
PROPELLANT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

PICSB-CW 15-Mar-93

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Minimum	mg/kg	USEPA, 1991
INGESTION RATE	IR	480	mg/day	Assumption
FRACTION INGESTED	FI	100%		USEPA, 1992
SOIL ADHERENCE FACTOR	SAF	1	mg/cm <sup>2</sup>	USEPA, 1990
SURFACE AREA EXPOSED	SA	2,100	cm <sup>2</sup> /day	USEPA, 1991
CONVERSION FACTOR	CF	0.000001	kg/mg	USEPA, 1991
BODY WEIGHT	BW	70	kg	USEPA, 1991
EXPOSURE FREQUENCY	EF	20	days/yr	USEPA, 1991
EXPOSURE DURATION	ED	1	years	USEPA, 1991
AVERAGING TIME	AT	70	years	USEPA, 1969
RELATIVE ABSORPTION FACTOR	RAF	0.0547945205	years	USEPA, 1991
CANCER				
NONCANCER				
INGESTION				
DERMAL				
Note: For noncardiogenic effects: AT = EF 365 days				
EQUATIONS: CANCER RISK = INTAKE (mg/kg-day) x CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup> HAZARD QUOTIENT = INTAKE (mg/kg-day) / REFERENCE DOSE (mg/kg-day) INTAKE = (INTAKE-INGESTION) + (INTAKE-DERMAL) INTAKE-INGESTION = $\frac{CS \times IR \times RAF \times FI \times CF \times EF \times ED}{BW \times AT \times 365 \text{ days/yr}}$ INTAKE-DERMAL = $\frac{CS \times SA \times SAF \times RAF \times CF \times EF \times ED}{BW \times AT \times 365 \text{ days/yr}}$				
USEPA, 1991. Risk Assessment Guidelines for Superfund USEPA, 1990. Exposure Factors Handbook USEPA, 1991. Standard Default Exposure Factors				
USEPA, 1992. Dermal Exposure Guidance				

TABLE O-7A, continued

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0-12 feet)

CONSTRUCTION WORKER

PROPELLANT BURNING GROUND

BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAI	INTAKE INGESTION (mg/kg-day)	DERMAL RAI	INTAKE DERMAL (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK DERMAL	TOTAL CANCER RISK
24DNT	58.9	1	3.2E-07	No Values Available for Quantitative Analysis		6.8E-01	2.1E-07		2.1E-07
24DNT	2	1	1.1E-08			6.8E-01	7.3E-09		7.3E-09
AS	18.8	1	1.0E-07			1.8E+00	1.8E-07		1.8E-07
B2EIP	6.2	1	3.3E-08			1.4E-02	4.7E-10		4.7E-10
BAA/NT	8.9	1	4.8E-08			7.3E+00	3.5E-07		3.5E-07
BAPYR	3.55	1	1.9E-08			7.3E+00	1.4E-07		1.4E-07
BEFANT	3.91	1	2.1E-08			7.3E+00	1.5E-07		1.5E-07
BEFANT	3.36	1	1.8E-08			7.3E+00	1.3E-07		1.3E-07
CB16	9.09	1	4.9E-08			2.9E-02	1.4E-09		1.4E-09
CIERY	8.28	1	4.4E-08			7.3E+00	3.2E-07		3.2E-07
CR	40.4	1	2.2E-07			ND			
DBA/1A	0.661	1	3.5E-09			7.3E+00	2.6E-08		2.6E-08
KCDPYR	4.52	1	2.4E-08			7.3E+00	1.8E-07		1.8E-07
NNDPA	12	1	6.4E-08			7.0E+00	4.5E-07		4.5E-07
PB	1200	1	6.4E-06			ND			
TCOLB	0.23	1	1.2E-09			1.1E-02	1.4E-11		1.4E-11
SUMMARY CANCER RISK							2E-06	8E+00	2E-06

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAP	INTAKE INGESTION (mg/kg-day)	DERMAL RAP	INTAKE DERMAL (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT DERMAL	TOTAL HAZARD QUOTIENT
24DNT	58.9	1	4.0E-04	No Values Available for Quantitative Analysis		2.0E-03	2.02E-01		2.02E-01
24DNT	2	1	1.4E-05			ND			
24NAP	18.2	1	1.2E-04			4.0E-02	3.12E-03		3.12E-03
ANAPNE	16.9	1	1.2E-04			4.0E-02	2.90E-03		2.90E-03
ANAPLY	1.04	1	7.1E-06			4.0E-02	1.78E-04		1.78E-04
ANTRC	12.4	1	8.5E-05			4.0E-02	2.13E-03		2.13E-03
AS	18.8	1	1.3E-04			3.0E-04	4.30E-01		4.30E-01
B2BIP	6.2	1	4.3E-05			2.0E-02	2.13E-03		2.13E-03
BAANTR	8.9	1	6.1E-05			4.0E-02	1.53E-03		1.53E-03
BAPTR	3.55	1	2.4E-05			4.0E-02	6.09E-04		6.09E-04
BEFANT	3.91	1	2.7E-05			4.0E-02	6.70E-04		6.70E-04
BCHPYP	2.57	1	1.8E-05			4.0E-02	4.41E-04		4.41E-04
BEFANT	3.36	1	2.3E-05			4.0E-02	5.76E-04		5.76E-04
CSH6	9.09	1	6.2E-05			ND			
CHRY	8.28	1	5.7E-05			4.0E-02	1.42E-03		1.42E-03
CR	40.4	1	2.8E-04			5.0E-03	5.54E-02		5.54E-02
CU	327.19	1	2.2E-03			ND			
DRAIA	0.661	1	4.5E-06			4.0E-02	1.13E-04		1.13E-04
DEZPUR	5.8	1	4.0E-05			ND			
DEP	6.2	1	4.3E-05			8.0E-01	5.31E-05		5.31E-05
DNDP	6.2	1	4.3E-05			1.0E-01	4.25E-04		4.25E-04
FANT	6.2	1	4.3E-05			4.0E-02	1.06E-03		1.06E-03
FLRENE	18.4	1	1.3E-04			4.0E-02	3.15E-03		3.15E-03
KDPYR	4.52	1	3.1E-05			4.0E-02	7.75E-04		7.75E-04
MCHSIS	14.4	1	9.9E-05			0.2	4.94E-04		4.94E-04
NAP	6.2	1	4.3E-05			0.04	1.06E-03		1.06E-03
NT	35	1	2.4E-04			0.1	2.40E-03		2.40E-03
NNDPA	12	1	8.2E-05			ND			
PS	1200	1	8.2E-03			ND			

TABLE O-7A, continued  
DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0-12 feet)  
CONSTRUCTION WORKER  
PROPELLANT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION		INTAKE INGESTION (mg/kg-day)	DERMAL RAP	INTAKE DERMAL (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT		TOTAL HAZARD QUOTIENT
		RAP						INGESTION	DERMAL	
PILANTR	12	1		8.2E-05			0.04	2.06E-03		2.06E-03
PYR	6.2	1		4.3E-05			0.03	1.42E-03		1.42E-03
SE	1.77	1		1.2E-05			0.005	2.43E-03		2.43E-03
SO4	280	1		1.9E-03			ND			
TRCLE	0.23	1		1.6E-06			ND			
TKLEN	39.5	1		2.7E-04			2	1.35E-04		1.35E-04
ZN	1253.94	1		8.6E-03			0.2	4.30E-02		4.30E-02
SUMMARY HAZARD INDEX										0.8

TABLE O-7B

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0-12 feet)  
CONSTRUCTION WORKER  
PROPELLANT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

PKCSHJOT 15-Mar-93

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Maximum	mg/kg	USEPA, 1991
INGESTION RATE	IR	480	mg/day	Assumption
FRAC TION INGESTED	FI	100%		USEPA, 1992
SOIL ADHERENCE FACTOR	SAF	1	mg/cm <sup>2</sup>	USEPA, 1990
SURFACE AREA EXPOSED	SA	2,100	cm <sup>2</sup> /day	USEPA, 1991
CONVERSION FACTOR	CF	0.000001	kg/mg	USEPA, 1991
BODY WEIGHT	BW	70	kg	USEPA, 1991
EXPOSURE FREQUENCY	EF	20	days/year	USEPA, 1991
EXPOSURE DURATION	ED	1	years	USEPA, 1991
AVERAGING TIME	AT	70	years	USEPA, 1989
RELATIVE ABSORPTION FACTOR	RAF	0.0547945203	years	USEPA, 1991
		1	unitless	USEPA, 1989

CANCER	$\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$
NONCANCER	$\text{HAZARD QUOTIENT} = \text{INTAKE (mg/kg-day)} / \text{REFERENCE DOSE (mg/kg-day)}$
INGESTION	$\text{INTAKE} = (\text{INTAKE-INGESTION}) + (\text{INTAKE-DERMAL})$
DERMAL	$\begin{aligned} \text{INTAKE-INGESTION} &= \frac{\text{CS} \times \text{IR} \times \text{RAF} \times \text{FI} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}} \\ \text{INTAKE-DERMAL} &= \frac{\text{CS} \times \text{SA} \times \text{SAF} \times \text{RAF} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}} \end{aligned}$

Note:  
For noncardiogenic effects: AT = EF / 365 days

USEPA, 1989, Risk Assessment Guidance for Superfund  
USEPA, 1990, Exposure Factors Handbook  
USEPA, 1991, Standard Default Exposure Factors

USEPA, 1992, Dermal Exposure Guidance

TABLE O-7B, continued  
DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0-12 feet)  
CONSTRUCTION WORKER  
PROPELLANT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAF	INTAKE INGESTION (mg/kg-day)	DERMAL RAF	INTAKE DERMAL (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK DERMAL	TOTAL CANCER RISK
24DNT	73000	1	3.9E-04	No Values Available for Quantitative Analysis		6.8E-01	2.7E-04		2.7E-04
SUMMARY CANCER RISK									3E-04
									0E+00
									3E-04

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAF	INTAKE INGESTION (mg/kg-day)	DERMAL RAF	INTAKE DERMAL (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT DERMAL	TOTAL HAZARD QUOTIENT
24DNT	73000	1	5.0E-01	No Values Available for Quantitative Analysis		2.0E-03	2.50E+02		2.50E+02
SUMMARY HAZARD INDEX									250.3
									0.9
									250.3



TABLE O-8  
INITIALATION EXPOSURE TO AMBIENT AIR  
CONSTRUCTION WORKER  
PROPELLANT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS

EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Maximum	mg/kg	0 zero - 12 feet
CONCENTRATION AIR PARTICULATES	CAp	Calculated	mg/m <sup>3</sup>	see below
CONCENTRATION AIR VOLATILES	CAv	Calculated	mg/m <sup>3</sup>	see below
VOLATILIZATION FACTOR	VF	Calculated	m <sup>3</sup> /kg	Appendix M
24 HOUR AVERAGE PM10 STANDARD	PM10	150	µg/m <sup>3</sup>	USEPA, 1991b
CONVERSION FACTOR	CF	1E-09	kg/kg	
INITIALATION RATE	IhR	2.5	m <sup>3</sup> /hour	USEPA, 1991a
BODY WEIGHT	BW	70	kg	USEPA, 1989
EXPOSURE TIME	ET	8	hour/day	Assumption
EXPOSURE FREQUENCY	EF	20	days/year	Assumption
EXPOSURE DURATION	ED	1	years	Assumption
AVERAGING TIME	AT	70	years	USEPA, 1991a
CANCER	AT	0.0540745205	years	USEPA, 1991a
NONCANCER	AT		years	

USEPA, 1989 Risk Assessment Guidelines for Superfund, Part A  
 USEPA, 1991a. Standard Default Exposure Factors  
 USEPA, 1991b. CFB, 50490-07

CANCER RISK = INTAKE (mg/kg-day) × CANCER SLOPE FACTOR (mg/kg-day)<sup>-1</sup> - 1  
 HAZARD QUOTIENT = AIR CONCENTRATION (mg/m<sup>3</sup>) / REFERENCE CONCENTRATION (mg/m<sup>3</sup>)  
 INTAKE =  $\frac{(CAp + CAv) \times IHR \times ET \times EF \times ED}{BW \times AT} \times CF$   
 AIR CONCENTRATION (mg/m<sup>3</sup>) = CAp + CAv  
 CAp = CS × PM10 × CF  
 CAv = CS × I/VF

Note:  
 For noncarcinogenic effects: AT = 365 days

TABLE O-8, continued  
INITIALATION EXPOSURE TO AMBIENT AIR  
CONSTRUCTION WORKER  
PROPELLANT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VF ( $\mu\text{g}/\text{kg}$ )	AIR CONCENTRATION VOLATILES ( $\mu\text{g}/\text{m}^3$ )	AIR CONCENTRATION PARTICULATES ( $\mu\text{g}/\text{m}^3$ )	INTAKE ( $\text{mg}/\text{kg}/\text{day}$ )	CANCER SLOPE FACTOR ( $\text{mg}/\text{kg}/\text{day}$ ) <sup>-1</sup>	CANCER RISK
2,4-DNT	58.9			0.00000635	2.0E-09	ND	
2,6-DNT	2			0.0000003	6.7E-11	ND	
AS	18.8			0.0000282	6.3E-10	5.0E+01	3.2E-06
BZBP	6.2			0.0000009	2.1E-10	ND	
CB6	9.09	4220	0.0021540284	0.00001365	4.8E-07	2.9E-02	1.4E-06
CR	40.4			0.00000608	1.4E-09	4.1E+01	5.6E-06
INDPA	12			0.0000018	4.0E-10	ND	
BAANTR	8.9			0.00001335	3.0E-10	6.1E+00	1.8E-09
BAPTR	3.55			0.00000325	1.2E-10	6.1E+00	7.3E-10
BEPANT	3.91			0.00000365	1.3E-10	6.1E+00	6.0E-10
BEPANT	3.36			0.00000304	1.1E-10	6.1E+00	6.9E-10
CBY	8.28			0.00001342	2.8E-10	6.1E+00	1.7E-09
DIABA	0.661			0.000000992	2.2E-11	6.1E+00	1.4E-10
ICPTR	4.52			0.00000678	1.5E-10	6.1E+00	9.2E-10
PS	1200			0.00018	4.0E-08	ND	
TRCCL	0.23			0.00000045	7.7E-12	1.7E-02	1.3E-13
SUMMARY CANCER RISK							1E-07

TABLE O-4, continued  
 INHALATION EXPOSURE TO AMBIENT AIR  
 CONSTRUCTION WORKER  
 PROPELLANT BURNING GROUND  
 BADGER ARMY AMMUNITION PLANT

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VP (m³/kg)	AIR CONCENTRATION VOLATILES (mg/m³)	AIR CONCENTRATION PARTICULATES (mg/m³)	REFERENCE CONCENTRATION (mg/kg.m)	HAZARD QUOTIENT VOLATILES	HAZARD QUOTIENT PARTICULATES	HAZARD QUOTIENT TOTAL
MERCURIES	1.04	8010	0.0001298177	0.000000156	2.0E+00	6.5E-03	7.8E-06	6.5E-03
No other COCs have MDCs.								
SUMMARY HAZARD INDEX						0.00006492	0.00000008	0.00006500

TABLE O-8A

INITIALATION EXPOSURE TO AMBIENT AIR  
CONSTRUCTION WORKER  
PROPELLANT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

PBGARW 25-Mar-95

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Maximum	mg/kg	@ zero - 12 feet
CONCENTRATION AIR PARTICULATES	CAp	Calculated	mg/m <sup>3</sup>	see below
CONCENTRATION AIR VOLATILES	CAv	Calculated	mg/m <sup>3</sup>	see below
VOLATILIZATION FACTOR	VF	Calculated	m <sup>3</sup> /kg	Appendix M
24 HOUR AVERAGE PM10 STANDARD	PM10	150	ug/m <sup>3</sup>	USEPA, 1991b
CONVERSION FACTOR	CF	1E-09	kg/ug	
INITIALATION RATE	IhR	2.5	m <sup>3</sup> /hour	USEPA, 1991a
BODY WEIGHT	BW	70	kg	USEPA, 1989
EXPOSURE TIME	ET	8	hours/day	Assumption
EXPOSURE FREQUENCY	EF	20	days/year	Assumption
EXPOSURE DURATION	ED	1	years	Assumption
AVERAGE TIME	AT	70	years	USEPA, 1991a
	AT	0.054794505	years	USEPA, 1991a
CANCER				
NONCANCER				

CANCER RISK = INTAKE (mg/kg-day) x CANCER SLOPE FACTOR (mg/kg-day)<sup>-1</sup> - 1  
 HAZARD QUOTIENT = AIR CONCENTRATION (mg/m<sup>3</sup>) / REFERENCE CONCENTRATION (mg/m<sup>3</sup>)  
 INTAKE = 
$$\frac{(CAp + CAv) \times IhR \times ET \times EF \times ED}{BW \times AT \times 365 \text{ days/yr}}$$
  
 AIR CONCENTRATION (mg/m<sup>3</sup>) = CAp + CAv  
 CAp = CS x PM10 x CF  
 CAv = CS x IVF

Note:  
 For noncarcinogenic effects: AT = EF 365 days

USEPA, 1989: Risk Assessment Guidance for Superfund, Part A  
 USEPA, 1991a: Standard Default Exposure Factors  
 USEPA, 1991b: CRELSD693-87

TABLE O-8A, continued  
 INHALATION EXPOSURE TO AMBIENT AIR  
 CONSTRUCTION WORKER  
 PROPELLANT BURNING GROUND  
 BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VP (m <sup>3</sup> /kg)	AIR CONCENTRATION VOLATILES (mg/m <sup>3</sup> )	AIR CONCENTRATION PARTICULATES (mg/m <sup>3</sup> )	INTAKE (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK
2,4-DNT	58.9			0.00000685	2.0E-09	ND	
2,6-DNT	2			0.0000003	6.7E-11	ND	
AS	18.8			0.00000282	6.3E-10	5.0E+01	3.2E-08
BZDHP	6.2			0.00000099	2.1E-10	ND	
CBZK	9.09			0.000001365	4.8E-07	2.9E-02	1.4E-08
CE	40.4	4220	0.0021540284	0.00000608	1.4E-09	4.1E+01	5.6E-08
MDPA	12			0.0000018	4.0E-10	ND	
BAANTH	8.9			0.000001335	3.0E-10	6.1E+00	1.8E-09
BAFTR	3.55			0.000000325	1.2E-10	6.1E+00	7.3E-10
BEFANT	3.91			0.000000865	1.3E-10	6.1E+00	8.0E-10
BEFANT	3.36			0.000000594	1.1E-10	6.1E+00	6.9E-10
CBZK	8.28			0.000001242	2.8E-10	6.1E+00	1.7E-09
DBABA	0.661			0.000000992	2.2E-11	6.1E+00	1.4E-10
NDPFR	4.52			0.000000678	1.5E-10	6.1E+00	9.2E-10
PS	1200			0.00018	4.0E-08	ND	
TRCLE	0.23			0.00000045	7.7E-12	1.7E-02	1.3E-13
SUMMARY CANCER RISK							1E-07

TABLE O-8A, continued  
INITIALATION EXPOSURE TO AMBIENT AIR  
CONSTRUCTION WORKER  
PROPELLANT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VF (m³/kg)	AIR CONCENTRATION		REFERENCE CONCENTRATION (mg/m³)	HAZARD QUOTIENT		HAZARD QUOTIENT TOTAL
			VOLATILES (mg/m³)	PARTICULATES (mg/m³)		VOLATILES	PARTICULATES	
MERCURY	14.4	8010	0.0017977526	0.00000216	2.0E+00	9.0E-04	1.1E-06	9.0E-04
No other COCs have RfCs								
SUMMARY HAZARD INDEX								
						0.000000000	0.000001000	0.000000000

TABLE B-8B  
INITIALATION EXPOSURE TO AMBIENT AIR  
CONSTRUCTION WORKER  
PROPELLANT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

EQUATIONS

EXPOSURE PARAMETERS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Median	mg/kg	0 zero - 12 feet
CONCENTRATION AIR PARTICULATES	CAP	Calculated	mg/m <sup>3</sup>	see below
CONCENTRATION AIR VOLATILES	CAV	Calculated	mg/m <sup>3</sup>	see below
VOLATILIZATION FACTOR	VF	Calculated	mg/m <sup>3</sup>	Appendix M
24 HOUR AVERAGE PM10 STANDARD	PM10	150	ug/m <sup>3</sup>	USEPA, 1991b
CONVERSION FACTOR	CF	1E-09	kg/ug	
INITIALATION RATE	IR	2.5	m <sup>3</sup> /hour	USEPA, 1991a
BODY WEIGHT	BW	70	kg	USEPA, 1989
EXPOSURE TIME	ET	8	hour/day	Assumption
EXPOSURE FREQUENCY	EF	20	day/year	Assumption
EXPOSURE DURATION	ED	1	years	Assumption
AVERAGE TIME	AT	70	years	USEPA, 1991a
CANCER	AT	0.054794505	years	USEPA, 1991a
NONCANCER	AT			

CANCER RISK = INTAKE (mg/kg-day) x CANCER SLOPE FACTOR (mg/kg-day)<sup>-1</sup> - 1  
 HAZARD QUOTIENT = AIR CONCENTRATION (mg/m<sup>3</sup>) / REFERENCE CONCENTRATION (mg/m<sup>3</sup>)  
 INTAKE = (CAP + CAV) x IR x ET x EF x ED  
 BW x AT x 365 day/yr  
 AIR CONCENTRATION (mg/m<sup>3</sup>) = CAP + CAV  
 CAP = CS x PM10 x CF  
 CAV = CS x 1/VF

Note:  
 For noncarcinogenic effects: AT = 365 days

TABLE O-8B, continued  
INITIALATION EXPOSURE TO AMBIENT AIR  
CONSTRUCTION WORKER  
PROPELLANT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VF (m³/kg)	AIR CONCENTRATION VOLATILES (mg/m³)	AIR CONCENTRATION PARTICULATES (mg/m³)	INTAKE (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day)⁻¹	CANCER RISK
2-MNT	73000			0.01095	2.4E-06	ND	
SUMMARY CANCER RISK							0E+00

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VF (m³/kg)	AIR CONCENTRATION VOLATILES (mg/m³)	AIR CONCENTRATION PARTICULATES (mg/m³)	REFERENCE CONCENTRATION (mg/m³)	HAZARD QUOTIENT VOLATILES	HAZARD QUOTIENT PARTICULATES	HAZARD QUOTIENT TOTAL
2-MNT	73000	8010	9.11360799	0.01095	ND			
SUMMARY HAZARD INDEX							0.00000000	0.00000000



**Table O-9**  
**Compounds Detected**  
**Final Creek Outflow**  
**Settling Ponds and Spoils T Area Surface Soil (0-2')**  
**U**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment (Y/N)?</u>	<u>Reason *</u>	<u>Exposure Point Concentration **</u>
AL	1: 1	21600		N	1	
ANAPYL	1: 1	0.166		Y		0.166
AS	1: 1	4.14		N	1	
B2EHP	1: 1	1.02		Y		1.02
BA	1: 1	183		N	1	
BAANTR	1: 1	0.185		Y		0.185
BBFANT	1: 1	0.723		Y		0.723
BE	1: 1	0.813		N	1	
BGHPY	1: 1	0.618		Y		0.618
BKFANT	1: 1	0.635		Y		0.635
CA	1: 1	6060		N	3	
CHRY	1: 1	0.264		Y		0.264
CO	1: 1	8.48		N	1	
CR	1: 1	23.9		N	1	
CU	1: 1	13.1		N	1	
FANT	1: 1	0.407		Y		0.407
FE	1: 1	23300		N	1, 3	
HG	1: 1	0.505		Y		0.505
K	1: 1	2340		N	1, 3	
MG	1: 1	3570		N	1, 3	
MN	1: 1	798		N	1	
NA	1: 1	79.3		N	1, 3	
NI	1: 1	15.9		N	1	
NIT	1: 1	3.53		Y		3.53
PB	1: 1	18		N	1	
PHANTR	1: 1	0.173		Y		0.173
PYR	1: 1	0.487		Y		0.487
SO4	1: 1	18.20		Y		18.20
TL	1: 1	2.14		N	1	
V	1: 1	62		N	1	
ZN	1: 1	67		N	1	

**Footnotes:**

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential for human nutrition.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum.

**Note:** Assessment of surface soil contamination (0 to 2 feet) was performed using data from boring SPB-91-01.

**Table O-10**  
**Compounds Detected**  
**Final Creek Outflow**  
**Settling Ponds and Spoils Disposal Area Subsurface Soil (2'-12')**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	<u>Concentration **</u>
AL	2 : 2	8050	2710	N	1	
BA	2 : 2	114	51.3	N	1	
CA	2 : 2	2130	1180	N	1, 3	
CO	2 : 2	11.8	3.33	N	1	
CR	2 : 2	10.9	7.43	N	1	
CU	2 : 2	6.85	4.87	N	1	
FE	2 : 2	13900	7180	N	3	
K	2 : 2	707	485	N	1, 3	
MG	2 : 2	1310	1050	N	1, 3	
MN	2 : 2	1300	1090	N	1	
NI	2 : 2	7.22	5.94	N	1	
NIT	2 : 2	3.76	3.46	Y		3.76
PB	2 : 2	10	2.15	N	1	
SO4	2 : 2	35.8	12.8	Y		35.8
TL	2 : 2	16.5	17	N	1	
V	2 : 2	46	14	N	1	
ZN	2 : 2	28	15	N	1	

**Footnotes:**

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential for human nutrition.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum

**Note:** Assessment of subsurface soil contamination (2 to 12 feet)  
was performed using data from boring SPB-91-01.

TABLE O-11

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
FINAL CREEK OUTFLOW AREA  
BADGER ARMY AMMUNITION PLANT

FOCUS336 25-Mar-93

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS		mg/kg	USEPA, 1991
INGESTION RATE - ADULT	IRa	100	mg/day	USEPA, 1991
INGESTION RATE - CHILD	IRc	200	mg/day	USEPA, 1991
PLACENT INGESTION	FI	100%		Assumption
CONVERSION FACTOR	CF	0.000001	kg/mg	USEPA, 1991
BODY WEIGHT - ADULT	BW <sub>a</sub>	70	kg	USEPA, 1991
BODY WEIGHT - CHILD	BW <sub>c</sub>	15	kg	USEPA, 1991
EXPOSURE FREQUENCY	EF	350	days/year	USEPA, 1991
EXPOSURE DURATION - ADULT	ED <sub>a</sub>	24	years	USEPA, 1991
EXPOSURE DURATION - CHILD	ED <sub>c</sub>	6	years	USEPA, 1991
AVERAGING TIME				
CANCER	AT	70	years	USEPA, 1999
ADULT - NONCANCER	AT <sub>a</sub>	24	years	USEPA, 1991
CHILD - NONCANCER	AT <sub>c</sub>	6	years	USEPA, 1991
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	USEPA, 1999

USEPA, 1999. Risk Assessment Guidelines for Superfund

USEPA, 1991. Standard Default Exposure Factors

Note:

Per contaminant exposure: AT = ED

CANCER RISK = INTAKE (mg/kg-day) x CANCER SLOPE FACTOR (mg/kg-day)<sup>-1</sup>

HAZARD QUOTIENT = INTAKE (mg/kg-day) / REFERENCE DOSE (mg/kg-day)

INTAKE-ADULT =

$$\frac{CS \times IRa \times FI \times CF \times EF \times ED_a}{BW_a \times AT_a \times 365 \text{ days/yr}}$$

INTAKE-CHILD =

$$\frac{CS \times IRc \times FI \times CF \times EF \times ED_c}{BW_c \times AT_c \times 365 \text{ days/yr}}$$

TABLE O-11, continued  
INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
FINAL CREEK OUTFLOW AREA  
BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAT	INTAKE ADULT (mg/kg-dw)	INTAKE CHILD (mg/kg-dw)	CANCER SLOPE FACTOR (mg/kg-dw) <sup>-1</sup>	CANCER RISK ADULT	CANCER RISK CHILD	TOTAL CANCER RISK
PCP	1.02	1	4.8E-07	1.1E-06	1.4E-02	6.7E-09	1.4E-08	2.2E-08
BAATHA	0.185	1	8.7E-08	2.0E-07	7.5E+00	6.5E-07	1.5E-06	2.1E-06
INFANT	0.775	1	3.4E-07	7.9E-07	7.5E+00	2.5E-06	5.8E-06	8.3E-06
INFANT	0.635	1	3.0E-07	7.0E-07	7.5E+00	2.2E-06	5.1E-06	7.3E-06
CHRY	0.264	1	1.2E-07	2.9E-07	7.5E+00	9.1E-07	2.1E-06	3.0E-06
PS	18	1	8.5E-06	2.0E-05	ND			
SUMMARY CANCER RISK								
						6E-06	1E-05	2E-05

TABLE O-11, continued

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
FINAL CREEK OUTFLOW AREA  
BADGER ARMY AMMUNITION PLANT

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAI	INTAKE ADULT (mg/kg-day)	INTAKE CHILD (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT ADULT	HAZARD QUOTIENT CHILD	TOTAL HAZARD QUOTIENT
ANATL	0.166	1	2.3E-07	2.1E-06	4.0E-02	5.68E-05	5.31E-05	5.97E-05
BZEP	1.02	1	1.4E-06	1.3E-05	2.0E-02	6.99E-05	6.52E-04	7.22E-04
BAANTR	0.185	1	2.5E-07	2.4E-06	4.0E-02	6.34E-05	5.91E-05	6.55E-05
BBFANT	0.723	1	9.9E-07	9.3E-06	4.0E-02	2.48E-05	2.31E-04	2.56E-04
BGRFP	0.618	1	8.5E-07	7.9E-06	4.0E-02	2.12E-05	1.98E-04	2.19E-04
BGFANT	0.635	1	8.7E-07	8.1E-06	4.0E-02	2.17E-05	2.05E-04	2.25E-04
CBRY	0.264	1	3.6E-07	3.4E-06	4.0E-02	9.04E-05	8.44E-05	9.34E-05
PANT	0.407	1	5.6E-07	5.2E-06	4.0E-02	1.39E-05	1.30E-04	1.44E-04
NO	0.505	1	6.9E-07	6.5E-06	3.0E-04	2.31E-05	2.15E-02	2.38E-02
MT	3.53	1	4.8E-06	4.5E-05	1.0E-01	4.84E-05	4.51E-04	5.00E-04
FB	18	1	2.5E-05	2.3E-04	ND			
PEANTR	0.173	1	2.4E-07	2.2E-06	4.0E-02	5.95E-05	5.53E-05	6.17E-05
PTR	0.487	1	6.7E-07	6.3E-06	3.0E-02	2.22E-05	2.08E-04	2.30E-04
SO4	18.2	1	2.5E-05	2.3E-04	ND			
SUMMARY HAZARD INDEX						0.0026	0.0035	0.0044

TABLE O-12

INCIDENTAL INGESTION AND INHALATION OF SOIL  
 GROUNDS MAINTENANCE WORKER  
 FINAL CREEK OUTFLOW AREA  
 BADGER ARMY AMMUNITION PLANT

FOUO SOW 08-Dec-92

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Median	mg/kg	USEPA, 1991a
INGESTION RATE	IR	100	mg/day	Assumption
FRACTION INGESTED	FI	100%		
CONVERSION FACTOR	CF	0.000001	kg/mg	
BODY WEIGHT	BW	70	kg	USEPA, 1991a
EXPOSURE FREQUENCY	EF	24	day/year	USEPA, 1991a
EXPOSURE DURATION	ED	25	years	USEPA, 1991a
CONCENTRATION AIR PARTICULATES	CAp	Calculated	mg/m <sup>3</sup>	Appendix M
CONCENTRATION AIR VOLATILES	CAv	Calculated	mg/m <sup>3</sup>	USEPA, 1991b
VOLATILIZATION FACTOR	VF	Calculated	m <sup>3</sup> /kg	USEPA, 1991a
PARTICULATE EMISSION FACTOR	PEF	4.03E+09	m <sup>3</sup> /hour	Assumption
RECALCULATION RATE	InH	2.5	hour/day	
EXPOSURE TIME	ET	8	hour/day	
AVERAGING TIME	AT	70	years	USEPA, 1989
	AT	25	years	USEPA, 1991a
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	USEPA, 1989

CANCER RISK = INTAKE (mg/kg-day) x CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	
HAZARD QUOTIENT <sub>ingestion</sub> = INTAKE (mg/kg-day) / REFERENCE DOSE (mg/kg-day)	
HAZARD QUOTIENT <sub>inhalation</sub> = $\frac{\text{AVERAGE AIR CONCENTRATION (mg/m}^3\text{)}}{\text{REFERENCE CONCENTRATION (mg/m}^3\text{)}}$	
INTAKE - INGESTION = $\frac{\text{CS} \times \text{IR} \times \text{RAF} \times \text{FI} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ day/yr}}$	
INTAKE - INHALATION = $\frac{(\text{CAp} + \text{CAv}) \times \text{InH} \times \text{ET} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ day/yr}}$	
AVERAGE AIR CONCENTRATION (mg/m <sup>3</sup> ) = CAp + CAv	
CAp = CS x 1/PEP	CAv = CS x 1/VP

Note:  
 For noncarcinogenic effects: AT = ED

USEPA, 1990. Risk Assessment Guidance for Superfund - Part A

USEPA, 1990. Exposure Factors Handbook

USEPA, 1991a. Standard Default Exposure Factors

USEPA, 1991b. Risk Assessment Guidance for Superfund - Part B

TABLE O-12, continued  
INCIDENTAL INGESTION AND INHALATION OF SOIL  
GROUNDS MAINTENANCE WORKER  
FINAL CREEK OUTFLOW AREA  
BADGER ARMY AMMUNITION PLANT

## CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAP	INTAKE INGESTION (mg/kg-day)	INTAKE INHALATION (mg/kg-day)	CANCER SLOPE FACTOR-DIRL (mg/kg-day) <sup>-1</sup>	CANCER SLOPE FACTOR-ING. (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK INHALATION	TOTAL CANCER RISK
B2E1P	1.02	1	3.4E-08	1.5E-12	ND	1.4E-02	4.8E-10		4.8E-10
BAANTR	0.183	1	6.2E-09	2.7E-13	6.1E+00	7.3E+00	4.5E-08	1.6E-12	4.5E-08
B2FANT	0.723	1	2.4E-08	1.0E-12	6.1E+00	7.3E+00	1.8E-07	6.4E-12	1.8E-07
B2FANT	0.635	1	2.1E-08	9.2E-13	6.1E+00	7.3E+00	1.6E-07	5.6E-12	1.6E-07
CHRY	0.264	1	8.9E-09	3.8E-13	6.1E+00	7.3E+00	6.5E-08	2.3E-12	6.5E-08
PB	18	1	6.0E-07	2.6E-11	ND	ND			
SUMMARY CANCER RISK									4E-07
									2E-11
									4E-07

INCIDENTAL INGESTION AND INHALATION OF SOIL  
 GROUNDS MAINTENANCE WORKER  
 FINAL CREEK OUTFLOW AREA  
 BADGER ARMY AMMUNITION PLANT

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION BAF	INGESTION (mg/kg-day)	AIR CONCENTRATION (mg/m <sup>3</sup> )	REFERENCE CONCENTRATION (mg/m <sup>3</sup> )	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT INHALATION	TOTAL HAZARD QUOTIENT
ANAPYL	0.166	1	1.6E-08	3.6E-11	ND	4.0E-02	3.90E-07		3.90E-07
B2EHP	1.02	1	9.6E-08	2.2E-10	ND	2.0E-02	4.79E-06		4.79E-06
BAACTR	0.185	1	1.7E-08	4.0E-11	ND	4.0E-02	4.34E-07		4.34E-07
BIFANT	0.723	1	6.8E-08	1.6E-10	ND	4.0E-02	1.70E-06		1.70E-06
BOHIPY	0.618	1	5.8E-08	1.3E-10	ND	4.0E-02	1.45E-06		1.45E-06
BKFANT	0.635	1	6.0E-08	1.4E-10	ND	4.0E-02	1.49E-06		1.49E-06
CHRY	0.264	1	2.5E-08	5.7E-11	ND	4.0E-02	6.20E-07		6.20E-07
FANT	0.407	1	3.8E-08	8.8E-11	ND	4.0E-02	9.56E-07		9.56E-07
HG	0.505	1	4.7E-08	1.1E-10	3.0E-04	3.0E-04	1.58E-04	3.6E-07	1.58E-04
NTT	3.53	1	3.3E-07	7.6E-10	ND	1.0E-01	3.32E-06		3.32E-06
PB	18	1	1.7E-06	3.9E-09	ND	ND			
PHANTR	0.173	1	1.6E-08	3.7E-11	ND	4.0E-02	4.06E-07		4.06E-07
PYR	0.487	1	4.6E-08	1.1E-10	ND	3.0E-02	1.52E-06		1.52E-06
SO4	18.2	1	1.7E-06	3.9E-09	ND	ND			
SUMMARY HAZARD INDEX							0.0002	0.0000	0.0002



TABLE O-13

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0-12 feet)  
CONSTRUCTION WORKER  
FINAL CREEK OUTFLOW AREA  
BADGER ARMY AMMUNITION PLANT

FOCUSB-CW 00-Dec-92

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Maximum	mg/kg	USEPA 1991
INGESTION RATE	IR	400	mg/day	Assumption
PLACATION EXPOSED	FI	100%		USEPA 1992
SOIL ADHERENCE FACTOR	SAF	1	mg/cm <sup>2</sup>	USEPA 1990
SURFACE AREA EXPOSED	SA	2,100	cm <sup>2</sup> /day	USEPA 1990
CONVERSION FACTOR	CF	0.00001	kg/kg	
BODY WEIGHT	BW	70	kg	USEPA 1991
EXPOSURE FREQUENCY	EF	20	days/year	USEPA 1991
EXPOSURE DURATION	ED	1	years	USEPA 1991
AVERAGING TIME	AT	70	years	USEPA 1989
RELATIVE ABSORPTION FACTOR	RAF	0.05-0.94 (200)	years	USEPA 1991
		1	unitless	USEPA 1989
		see (eq)		
CANCER RISK = INTAKE (mg/kg-day) x CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup> HAZARD QUOTIENT = INTAKE (mg/kg-day) / REFERENCE DOSE (mg/kg-day) INTAKE = (INTAKE-INGESTION) + (INTAKE-DERMAL) INTAKE-INGESTION = $\frac{CS \times IR \times SAF \times FI \times CF \times EF \times ED}{BW \times AT \times 365 \text{ days/yr}}$ INTAKE-DERMAL = $\frac{CS \times SA \times SAF \times RAF \times CF \times EF \times ED}{BW \times AT \times 365 \text{ days/yr}}$				
Note: For noncarcinogenic effects: AT = EF 365 days				
USEPA 1989. Risk Assessment Guidelines for Superfund				
USEPA 1990. Exposure Factors Handbook				
USEPA 1991. Standard Default Exposure Factors				
USEPA 1992. Dermal Exposure Guidelines				

TABLE O-13, continued  
DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0-12 feet)  
CONSTRUCTION WORKER  
FINAL CREEK OUTFLOW AREA  
BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAP	INGESTION (mg/kg-day)	DERMAL RAP	DERMAL INTAKE (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK DERMAL	TOTAL CANCER RISK
BENP	1.02	1	5.5E-09	No Values		1.4E-02	7.7E-11		7.7E-11
BAA/NT	0.185	1	9.9E-10	Available		7.3E+00	7.2E-09		7.2E-09
INFANT	0.723	1	3.9E-09	for		7.3E+00	2.8E-08		2.8E-08
INFANT	0.635	1	3.4E-09	Quasistative		7.3E+00	2.5E-08		2.5E-08
CHRY	0.264	1	1.4E-09	Analysis		7.3E+00	1.0E-08		1.0E-08
PB	18	1	9.7E-08			ND			
SUMMARY CANCER RISK									
							7E-08	9E+00	7E-08

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0-12 feet)

CONSTRUCTION WORKER

FINAL CREEK OUTFLOW AREA

BADGER ARMY AMMUNITION PLANT

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAI	INTAKE INGESTION (mg/kg-day)	DERMAL RAI	INTAKE DERMAL (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT DERMAL	TOTAL HAZARD QUOTIENT
ANAPYL	0.166	1	1.1E-06	No Values Available for Quantitative Analysis	4.0E-02	4.0E-02	2.85E-05		2.85E-05
BZEP	1.02	1	7.0E-06		2.0E-02	2.0E-02	3.50E-04		3.50E-04
BAANTR	0.185	1	1.3E-06		4.0E-02	4.0E-02	3.17E-05		3.17E-05
BEPANT	0.723	1	5.0E-06		4.0E-02	4.0E-02	1.24E-04		1.24E-04
BOHIPY	0.618	1	4.2E-06		4.0E-02	4.0E-02	1.06E-04		1.06E-04
BEPANT	0.635	1	4.4E-06		4.0E-02	4.0E-02	1.09E-04		1.09E-04
CHRY	0.264	1	1.8E-06		4.0E-02	4.0E-02	4.53E-05		4.53E-05
PANT	0.407	1	2.8E-06		4.0E-02	4.0E-02	6.98E-05		6.98E-05
IRG	0.505	1	3.5E-06		3.0E-04	3.0E-04	1.15E-02		1.15E-02
NET	3.76	1	2.6E-05		1.0E-01	1.0E-01	2.58E-04		2.58E-04
P8	18	1	1.2E-04		ND	ND			
PHANTR	0.173	1	1.2E-06		4.0E-02	4.0E-02	2.97E-05		2.97E-05
PYR	0.487	1	3.3E-06		3.0E-02	3.0E-02	1.11E-04		1.11E-04
SO4	35.8	1	2.5E-04		ND	ND			
SUMMARY HAZARD INDEX							0.01	0.00	0.01

TABLE O-14  
INITIALATION EXPOSURE TO AMBIENT AIR  
CONSTRUCTION WORKER  
FINDAL CREEK OUTFLOW AREA  
BADGER ARMY AMMUNITION PLANT

EQUATIONS

EXPOSURE PARAMETERS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Median	mg/kg	@ zero - 12 feet
CONCENTRATION AIR PARTICULATES	CAp	Calculated	mg/m <sup>3</sup>	see below
CONCENTRATION AIR VOLATILES	CAv	Calculated	mg/m <sup>3</sup>	see below
VOLATILIZATION FACTOR	VF	Calculated	m <sup>3</sup> /kg	Appendix M
24 HOUR AVERAGE PM10 STANDARD	PM10	150	µg/m <sup>3</sup>	USEPA, 1991b
CONVERSION FACTOR	CF	1E-09	kg/kg	USEPA, 1991a
IRRADIATION RATE	IR	2.5	m <sup>2</sup> /hour	USEPA, 1991a
BODY WEIGHT	BW	70	kg	USEPA, 1991a
EXPOSURE TIME	ET	8	hour/day	Assumption
EXPOSURE FREQUENCY	EF	20	day/year	Assumption
EXPOSURE DURATION	ED	1	years	Assumption
AVERAGING TIME	AT	70	years	USEPA, 1991a
CANCER	AT	0.0547945203	years	USEPA, 1991b
NONCANCER	AT			

CANCER RISK = INTAKE (mg/kg-day) × CANCER SLOPE FACTOR (mg/kg-day)<sup>-1</sup> - 1  
 HAZARD QUOTIENT = AIR CONCENTRATION (mg/m<sup>3</sup>) / REFERENCE CONCENTRATION (mg/m<sup>3</sup>)  
 INTAKE =  $\frac{(CAp + CAv) \times IR \times ET \times EF \times ED}{BW \times AT}$   
 AIR CONCENTRATION (mg/m<sup>3</sup>) = CAp + CAv  
 CAp = CS × PM10 × CF  
 CAv = CS × VVF

Note:  
 For noncarcinogenic effects: AT = EF  
 365 days

USEPA, 1991a, Risk Assessment Guidelines for Superfund, Part A  
 USEPA, 1991a, Standard Default Exposure Factors  
 USEPA, 1991b, CFS-50293-87

TABLE O-14, continued  
 INHALATION EXPOSURE TO AMBIENT AIR  
 CONSTRUCTION WORKER  
 FINAL CREEK OUTFLOW AREA  
 BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VF (m³/kg)	AIR CONCENTRATION VOLATILES (mg/m³)	AIR CONCENTRATION PARTICULATES (mg/m³)	BTAS (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day)⁻¹	CANCER RISK
BZEMP	1.02			0.000000153	3.4E-11	ND	
BAANTH	0.185			0.0000000278	6.2E-12	6.1E+00	3.8E-11
BEFANT	0.723			0.0000001085	2.4E-11	6.1E+00	1.5E-10
BEFANT	0.635			0.0000000933	2.1E-11	6.1E+00	1.3E-10
CBRY	0.264			0.0000000996	8.9E-12	6.1E+00	5.4E-11
FB	18			0.0000027	6.0E-10	ND	
SUMMARY CANCER RISK							4E-10

TABLE O-14, continued  
 INHALATION EXPOSURE TO AMBIENT AIR  
 CONSTRUCTION WORKER  
 FINAL CREEK OUTFLOW AREA  
 BADGER ARMY AMMUNITION PLANT

## NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VP (mmHg)	AIR CONCENTRATION VOLATILES (mg/m <sup>3</sup> )	AIR CONCENTRATION PARTICULATES (mg/m <sup>3</sup> )	REFERENCE CONCENTRATION (mg/m <sup>3</sup> )	HAZARD QUOTIENT VOLATILES	HAZARD QUOTIENT PARTICULATES	HAZARD QUOTIENT TOTAL
AKAPYL	0.166				ND			
BZEP	1.02			0.000000249	ND			
BAANTR	0.185			0.000000153	ND			
BEPANT	0.723			0.000000278	ND			
BGEOPY	0.618			0.000001083	ND			
BEPANT	0.635			0.000000927	ND			
CEBY	0.264			0.000000953	ND			
PAINT	0.407			0.000000896	ND			
EG	0.905			0.000000811	ND			
MT	3.76			0.000000758	8.8E-05		8.8E-04	8.8E-04
PS	18			0.000000564	ND			
FEANTR	0.173			0.0000027	ND			
PYR	0.487			0.000000266	ND			
SO4	33.8			0.000000731	ND			
				0.0000037	ND			
SUMMARY HAZARD INDEX						0.0000	0.0009	0.0009

**Table O-15**  
**Compounds Detected**  
**Final Creek**  
**Settling Ponds and Spoils Disposal Area Surface Soil (0-2')**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment (Y/N)?</u>	<u>Reason *</u>	<u>Exposure Point Concentration **</u>
AL	8 : 8	14000	890	N	1	
PB	8 : 8	40	3.6	Y		40
K	8 : 8	920	26	N	1, 3	
NA	8 : 8	180	18	N	1, 3	
SN	7 : 8	63	25	Y		63
NIT	8 : 8	11	1.6	Y		11
NH3	8 : 8	1800	24	Y		1800
SO4	4 : 8	260	28	Y		260
24DNT	5 : 8	6	0.17	Y		6
26DNT	6 : 8	40	1.6	Y		40
DEP	2 : 8	0.13	0.11	Y		0.13
DNBP	5 : 8	26	1.7	Y		26
DPA	6 : 8	15	0.22	Y		15
2NNDPA	3 : 8	2	0.57	Y		2
NC	3 : 8	740	100	Y		740

Footnotes: \* 1 = within background range.  
\* 2 = laboratory or sampling contaminant  
\* 3 = essential for human nutrition  
\* 4 = frequency of detection less than 5 %  
\*\* 95th percentile or maximum

Note: Assessment of surface soil contamination (0 to 2 feet) was performed using data from samples FC-1 through FC-8.

TABLE O-16

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
FINAL CREEK  
BADGER ARMY AMMUNITION PLANT

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Medians	mg/kg	USEPA 1991
INGESTION RATE - ADULT	IRa	100	mg/day	USEPA 1991
INGESTION RATE - CHILD	IRc	200	mg/day	USEPA 1991
FRACTION INGESTED	FI	100%		Assumption
CONVERSION FACTOR	CF	0.000001	kg/mg	USEPA 1991
BODY WEIGHT - ADULT	BWa	70	kg	USEPA 1991
BODY WEIGHT - CHILD	BWc	15	kg	USEPA 1991
EXPOSURE FREQUENCY	EF	350	day/year	USEPA 1991
EXPOSURE DURATION - ADULT	EDa	24	years	USEPA 1991
EXPOSURE DURATION - CHILD	EDc	6	years	USEPA 1991
AVERAGING TIME				
CANCER	AT	70	years	USEPA 1999
ADULT - NONCANCER	ATa	24	years	USEPA 1991
CHILD - NONCANCER	ATc	6	years	USEPA 1991
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	USEPA 1999

USEPA 1999. Risk Assessment Guidelines for Superfund  
USEPA 1991. Standard Default Exposure Factors

Notes:

Per noncarcinogenic effects: AT = ED

CANCER RISK = INTAKE (mg/kg-day) x CANCER SLOPE FACTOR (mg/kg-day)<sup>-1</sup>

HAZARD QUOTIENT = INTAKE (mg/kg-day) / REFERENCE DOSE (mg/kg-day)

INTAKE-ADULT =

$CS \times IRa \times FI \times CF \times EF \times EDa$   
BWa x ATa x 365 day/yr

INTAKE-CHILD =

$CS \times IRc \times FI \times CF \times EF \times EDc$   
BWc x ATc x 365 day/yr



TABLE O-14, continued

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
FINAL CREEK  
BADGER ARMY AMMUNITION PLANT

## CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RPF	INTAKE ADULT (mg/kg-dw)	INTAKE CHILD (mg/kg-dw)	CANCER SLOPE FACTOR (mg/kg-dw) <sup>-1</sup>	CANCER RISK ADULT	CANCER RISK CHILD	TOTAL CANCER RISK
2,4-DT	6	1	2.8E-06	6.8E-06	6.8E-01	1.9E-06	4.5E-06	6.4E-06
2,4-DT	40	1	1.9E-05	4.8E-05	6.8E-01	1.3E-05	3.0E-05	4.3E-05
FB	40	1	1.9E-05	4.8E-05	ND			
SUMMARY CANCER RISK						1E-05	3E-05	5E-05

TABLE O-14, continued

INCIDENTAL INJECTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
FINAL CREEK  
BADGER ARMY AMMUNITION PLANT

## NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION DAP	INTAKE (mg/kg-day)		REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT		HAZARD QUOTIENT CHILD	TOTAL HAZARD QUOTIENT
			ADULT	CHILD		ADULT	CHILD		
PB	40	1	5.5E-05	5.1E-04	ND				
SN	63	1	8.6E-05	8.1E-04	6.0E-01	1.4E-04	1.34E-03	1.49E-03	
MT	11	1	1.5E-05	1.4E-04	1.0E-01	1.51E-04	1.41E-03	1.56E-03	
NR3	1800	1	2.5E-03	2.5E-02	ND				
SO4	260	1	3.6E-04	3.5E-03	ND				
24DNT	6	1	8.2E-06	7.7E-05	2.0E-03	4.11E-03	3.84E-02	4.25E-02	
24DNT	40	1	5.5E-05	5.1E-04	ND				
DNBP	0.13	1	1.8E-07	1.7E-06	8.0E-01	2.23E-07	2.08E-06	2.50E-06	
DNBP	26	1	3.6E-05	3.5E-04	1.0E-01	3.56E-04	3.32E-03	3.68E-03	
DPA	15	1	2.1E-05	1.9E-04	2.5E-02	8.22E-04	7.67E-03	8.49E-03	
24NDPA	2	1	2.7E-06	2.6E-05	ND				
NC	740	1	1.0E-03	9.5E-03	ND				
SUMMARY HAZARD INDEX									0.052
									0.053

**TABLE O-17**  
**INCIDENTAL INGESTION AND INHALATION OF SOIL**  
**BY GROUNDS MAINTENANCE WORKER**  
**AT LEADGER ARMY AMMUNITION PLANT**  
**FINAL CREEK**

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Medium	mg/kg	USEPA, 1991a
INGESTION RATE	IR	100	mg/day	Assumption
FRACTION INGESTED	FI	100%		
CONVERSION FACTOR	CF	0.00001		
BODY WEIGHT	BW	70	kg	USEPA, 1991a
EXPOSURE FREQUENCY	EF	24	days/year	USEPA, 1991a
EXPOSURE DURATION	ED	25	years	USEPA, 1991a
CONCENTRATION AIR PARTICULATES	Cap	Calculated	mg/m <sup>3</sup>	Appendix M
CONCENTRATION AIR VOLATILES	CAV	Calculated	mg/m <sup>3</sup>	USEPA, 1991b
VOLATILIZATION FACTOR	VF	Calculated	mg/kg	USEPA, 1991a
PARTICULATE EMISSION FACTOR	PEF	4.63E-09	m <sup>3</sup> /hour	Assumption
INHALATION RATE	IRR	2.5	m <sup>3</sup> /hour	
EXPOSURE TIME	ET	8	hours/day	
AVERAGING TIME				
RELATIVE ABSORPTION FACTOR				
CANCER	AT	70	years	USEPA, 1990
NONCANCER	AT	25	years	USEPA, 1991a
	RAF	1	unitless	USEPA, 1990

CANCER RISK = INTAKE (mg/kg-day) × CANCER SLOPE FACTOR (mg/kg-day)<sup>-1</sup>

HAZARD QUOTIENT<sub>ingestion</sub> = INTAKE (mg/kg-day) / REFERENCE DOSE (mg/kg-day)

HAZARD QUOTIENT<sub>inhalation</sub> =  $\frac{\text{AIR CONCENTRATION (mg/m}^3\text{)}}{\text{REFERENCE CONCENTRATION (mg/m}^3\text{)}}$

INTAKE-INGESTION =  $\frac{\text{CS} \times \text{IR} \times \text{FI} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/year}}$

INTAKE-INHALATION =  $\frac{(\text{Cap} + \text{CAV}) \times \text{IRR} \times \text{ET} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/year}}$

AIR CONCENTRATION (mg/m<sup>3</sup>) = Cap + CAV

Cap = CS × UFER

CAV = CS × UVF

Note:

For cancer dosimetric effects: AT = ED

USEPA, 1990. Risk Assessment Guidelines for Superfund - Part A

USEPA, 1990. Exposure Factors Handbook

USEPA, 1991a. Standard Default Exposure Factors

USEPA, 1991b. Risk Assessment Guidelines for Superfund - Part B

**USHPA 1999. Risk Assessment Guidance for Superfund - Part A**

USEPA 1993 Exposure Factors Handbook

USEPA 1974. Standard Defect Exposure Factors

USEPA 1991b. Risk Assessment Guidelines for Superfund—Part B

**Note:**

**For noncardiovascular effects: AT = ED**

TABLE O-17, continued  
INCIDENTAL INGESTION AND INHALATION OF SOIL  
GROUNDS MAINTENANCE WORKER  
FINAL CREEK  
BADGER ARMY AMMUNITION PLANT

PCSSGW 08-Dec-92

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION BAF	INTAKE INGESTION (mg/kg-day)	INTAKE INHALATION (mg/kg-day)	CANCER SLOPE FACTOR-INEL (mg/kg-day) <sup>-1</sup>	CANCER SLOPE FACTOR-DNO (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK INHALATION	TOTAL CANCER RISK
24DNT	6	1	2.0E-07	8.7E-12	ND	6.8E-01	1.4E-07		1.4E-07
24DNT	40	1	1.3E-06	5.8E-11	ND	6.8E-01	9.1E-07		9.1E-07
PS	40	1	1.3E-06	5.8E-11	ND				
SUMMARY CANCER RISK									
							1E-06	9E+00	1E-06

TABLE O-17, continued  
INCIDENTAL INGESTION AND INHALATION OF SOIL  
GROUNDS MAINTENANCE WORKER  
FINAL CREEK  
BADGER ARMY AMMUNITION PLANT

## NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION BAF	INTAKE INGESTION (mg/kg-day)	AIR CONCENT. (mg/m <sup>3</sup> )	REFERENCE CONCENT. (mg/m <sup>3</sup> )	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT INHALATION	TOTAL HAZARD QUOTIENT
PB	40	1	3.8E-06	8.6E-09	ND	ND	9.86E-06		9.86E-06
SN	63	1	5.9E-06	1.4E-08	ND	6.0E-01	1.03E-05		1.03E-05
MT	11	1	1.0E-06	2.4E-09	ND	1.0E-01			
NB	1800	1	1.7E-04	3.9E-07	ND	ND			
SO4	260	1	2.4E-05	5.6E-08	ND	ND			
24DNT	6	1	5.6E-07	1.3E-09	ND	2.0E-03	2.82E-04		2.82E-04
26DNT	40	1	3.8E-06	8.6E-09	ND	ND			
DEP	0.13	1	1.2E-08	2.8E-11	ND	8.0E-01	1.53E-08		1.53E-08
DNSP	26	1	2.4E-06	5.6E-09	ND	1.0E-01	2.44E-05		2.44E-05
DPA	15	1	1.4E-06	3.2E-09	ND	2.0E-02	7.05E-05		7.05E-05
2NDPA	2	1	1.9E-07	4.3E-10	ND	ND			
NC	740	1	7.0E-05	1.6E-07	ND	ND			
SUMMARY HAZARD INDEX							0.0004	0.0000	0.0004

**Table O-18**  
**Compounds Detected**  
**Settling Pond 1**  
**Settling Ponds and Spoils Disposal Area Surface Soil (0-2')**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
AL	17 : 17	27000	1400	N	1	
PB	16 : 17	180	5.1	Y		180
K	14 : 14	1100	69	N	1, 3	
NA	14 : 14	150	17	N	1, 3	
SN	17 : 17	57	0.45	Y		57
NIT	14 : 16	13	0.2	Y		13
NH3	14 : 14	740	53	Y		740
SO4	8 : 18	2500	58	Y		2500
24DNT	5 : 15	172	0.03	Y		172
26DNT	6 : 14	26	0.16	Y		26
DEP	1 : 15	460		Y		460
DNBP	6 : 15	14	0.1	Y		14
DPA	6 : 14	10	0.24	Y		10
2NNDPA	3 : 14	0.97	0.72	Y		0.97
NC	7 : 15	60000	180	Y		60000

Footnotes:

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant
- \* 3 = essential for human nutrition
- \* 4 = frequency of detection less than 5 %
- \*\* 95th percentile or maximum

Note: Assessment of surface soil contamination (0 to 2 feet) was performed using data from samples FPI-1 through FPI-14, and S1201 through S1204.

**Table O-19**  
**Compounds Detected**  
**Settling Pond 1**  
**Settling Ponds and Spoils Disposal Area Subsurface Soil (2'-16')**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	<u>Concentration **</u>
DEP	3 : 4	1340	5	Y		1340
24DNT	4 : 4	17.1	0.087	Y		17.1

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant.  
                   \* 3 = essential for human nutrition.  
                   \* 4 = frequency of detection less than 5 %.  
                   \*\* 95th percentile or maximum.

Notes:       Assessment of subsurface soil contamination (2 to 16 feet)  
                   was performed using data from samples S1201 through S1204.

TABLE O-29

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SETTLING POND 1  
BADGER ARMY AMMUNITION PLANT

EP 183061

25-Mar-93

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	Cs	Medium	mg/kg	USEPA, 1991
INGESTION RATE - ADULT	IRa	100	mg/day	USEPA, 1991
INGESTION RATE - CHILD	IRc	200	mg/day	USEPA, 1991
FRACTION INGESTED	FI	100%		Assumption
CONVERSION FACTOR	CF	0.000001	kg/kg	USEPA, 1991
BODY WEIGHT - ADULT	BWa	70	kg	USEPA, 1991
BODY WEIGHT - CHILD	BWc	15	kg	USEPA, 1991
EXPOSURE FREQUENCY	EF	350	days/year	USEPA, 1991
EXPOSURE DURATION - ADULT	EDa	24	years	USEPA, 1991
EXPOSURE DURATION - CHILD	EDc	6	years	USEPA, 1991
AVERAGING TIME	AT	70	years	USEPA, 1989
CANCER	ATc	24	years	USEPA, 1991
ADULT - NONCANCER	ATc	6	years	USEPA, 1991
CHILD - NONCANCER	RAF	1	unitless	USEPA, 1989
RELATIVE ABSORPTION FACTOR				

USEPA, 1989. Risk Assessment Guidance for Superfund  
USEPA, 1991. Standard Default Exposure Factors

Note:

For noncarcinogenic effects: AT = ED

CANCER RISK = INTAKE (mg/kg-day) x CANCER SLOPE FACTOR (mg/kg-day)<sup>-1</sup>

HAZARD QUOTIENT = INTAKE (mg/kg-day) / REFERENCE DOSE (mg/kg-day)

INTAKE-ADULT =

$$\frac{Cs \times IRa \times FI \times CF \times EF \times EDa}{BWa \times ATa \times 365 \text{ days/yr}}$$

INTAKE-CHILD =

$$\frac{Cs \times IRc \times FI \times CF \times EF \times EDc}{BWc \times ATc \times 365 \text{ days/yr}}$$



TABLE O-28, continued  
INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SETTLING POND 1  
BADGER ARMY AMMUNITION PLANT

REV 183308 23-Mar-93

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAT	INTAKE ADULT (mg/kg-day)	INTAKE CHILD (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK ADULT	CANCER RISK CHILD	TOTAL CANCER RISK
240MT	172	1	8.1E-03	1.9E-04	6.8E-01	5.5E-03	1.3E-04	1.8E-04
280MT	26	1	1.2E-03	2.8E-03	6.8E-01	8.3E-06	1.9E-05	2.8E-05
79	180	1	8.3E-03	2.0E-04	ND			
SUMMARY CANCER RISK						6E-03	1E-04	2E-04

TABLE O-20, continued

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SETTLING POND 1  
BADGER ARMY AMMUNITION PLANT

## NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAT	INTAKE ADULT (mg/kg-day)	INTAKE CHILD (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT ADULT	HAZARD QUOTIENT CHILD	TOTAL HAZARD QUOTIENT
PB	180	1	2.5E-04	2.5E-03	ND			
SN	57	1	7.8E-05	7.3E-04	6.0E-01	1.30E-04	1.21E-03	1.34E-03
MTT	13	1	1.8E-05	1.7E-04	1.0E-01	1.78E-04	1.66E-03	1.84E-03
NEB	740	1	1.0E-03	9.5E-03	ND			
SO4	2500	1	3.4E-03	3.2E-02	ND			
24DNT	172	1	2.4E-04	2.2E-03	2.0E-03	1.18E-01	1.10E+00	1.22E+00
26DNT	26	1	3.6E-05	3.3E-04	ND			
DHP	460	1	6.3E-04	5.9E-03	8.0E-01	7.88E-04	7.55E-03	8.14E-03
DHPF	14	1	1.9E-05	1.8E-04	1.0E-01	1.92E-04	1.79E-03	1.98E-03
DFA	10	1	1.4E-05	1.3E-04	2.5E-02	5.48E-04	5.11E-03	5.64E-03
2,3,4,5-TCDF	0.97	1	1.3E-06	1.2E-05	ND			
PC	60000	1	8.2E-02	7.7E-01	ND			
SUMMARY HAZARD INDEX						0.12	1.12	1.24

TABLE O-21  
INCIDENTAL INGESTION AND INHALATION OF SOIL  
GROUNDS MAINTENANCE WORKER  
SETTLING POND 1  
BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS

EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Medium	mg/kg	USEPA, 1991a
INGESTION RATE	IR	100	mg/day	Assumption
FRACTION INGESTED	FI	100%		
CONVERSION FACTOR	CF	0.000001	kg/mg	USEPA, 1991a
BODY WEIGHT	BW	70	kg	USEPA, 1991a
EXPOSURE FREQUENCY	EF	24	days/year	USEPA, 1991a
EXPOSURE DURATION	ED	25	years	USEPA, 1991a
CONCENTRATION AIR PARTICULATES	CAp	Calculated	mg/m <sup>3</sup>	Approach M
VOLATILIZATION FACTOR	VF	Calculated	mg/m <sup>3</sup>	USEPA, 1991b
PARTICULATE EMISSION FACTOR	PEF	Calculated	m <sup>3</sup> /kg	USEPA, 1991a
WEALATION RATE	WR	4.63E+09	m <sup>3</sup> /hour	Assumption
EXPOSURE TIME	ET	2.5	hours/day	
AVERAGING TIME	AT	8	hours/day	
CANCER				
NONCANCER				
RELATIVE ABSORPTION FACTOR	RAF	1		

USEPA, 1991. Risk Assessment Guidance for Superfund - Part A

USEPA, 1990. Exposure Factors Handbook

USEPA, 1991a. Standard Default Exposure Factors

USEPA, 1991b. Risk Assessment Guidance for Superfund - Part B

$$\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$$

$$\text{HAZARD QUOTIENT}_{\text{Ingestion}} = \text{INTAKE (mg/kg-day)} / \text{REFERENCE DOSE (mg/kg-day)}$$

$$\text{HAZARD QUOTIENT}_{\text{Inhalation}} = \frac{\text{AIR CONCENTRATION (mg/m}^3\text{)}}{\text{REFERENCE CONCENTRATION (mg/m}^3\text{)}}$$

$$\text{INTAKE - INGESTION} = \frac{\text{CS} \times \text{IR} \times \text{FI} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

$$\text{INTAKE - INHALATION} = \frac{(\text{CAp} \times \text{CAv}) \times \text{IR} \times \text{FI} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

$$\text{AIR CONCENTRATION (mg/m}^3\text{)} = \text{CAp} \times \text{CAv}$$

$$\text{CAp} = \text{CS} \times \text{VF} \quad \text{CAv} = \text{CS} \times \text{VF} \times \text{RAF}$$

Note:

For noncarcinogenic effects: AT = ED

TABLE O-21, continued  
INCIDENTAL INGESTION AND INHALATION OF SOIL  
GROUNDS MAINTENANCE WORKER  
SETTLING POND 1  
BADGER ARMY AMMUNITION PLANT

SP1550W 06-Dec-92

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION DAF	INTAKE INGESTION (mg/kg-day)	INTAKE INHALATION (mg/kg-day)	CANCER SLOPE FACTOR-ING. (mg/kg-day) <sup>-1</sup>	CANCER SLOPE FACTOR-ING. (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK INHALATION	TOTAL CANCER RISK
24DNT	172	1	5.8E-06	2.5E-10	ND	6.8E-01	3.9E-06		3.9E-06
24DNT	26	1	8.7E-07	3.8E-11	ND	6.8E-01	5.9E-07		5.9E-07
PS	180	1	6.0E-06	2.6E-10	ND				
SUMMARY CANCER RISK									5E-06
									6E-06

TABLE O-21, continued

INCIDENTAL INGESTION AND INHALATION OF SOIL  
 GROUNDS MAINTENANCE WORKER  
 SETTLING POND 1  
 BADGER ARMY AMMUNITION PLANT

## NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION BAF	ENTREE INGESTION (mg/kg-day)	AIR CONCENT. (mg/m <sup>3</sup> )	REFERENCE CONCENT. (mg/m <sup>3</sup> )	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT INHALATION	TOTAL HAZARD QUOTIENT
PB	180	1	1.7E-05	3.9E-08	ND	ND	8.92E-06		8.92E-06
SN	57	1	5.4E-06	1.2E-08	ND	6.0E-01	1.22E-05		1.22E-05
NET	13	1	1.2E-06	2.8E-09	ND	1.0E-01			
NEIS	740	1	7.0E-05	1.4E-07	ND	ND			
SO4	2500	1	2.3E-04	5.4E-07	ND	ND			
24DNT	172	1	1.4E-05	3.7E-08	ND	2.0E-03	8.08E-03		8.08E-03
26DNT	26	1	2.4E-06	5.6E-09	ND	ND			
DEP	440	1	4.3E-05	9.9E-08	ND	8.0E-01	5.40E-05		5.40E-05
DNSP	14	1	1.3E-06	3.0E-09	ND	1.0E-01	1.32E-05		1.32E-05
DPA	10	1	9.4E-07	2.2E-09	ND	2.0E-02	4.70E-05		4.70E-05
24NDPA	0.97	1	9.1E-08	2.1E-10	ND	ND			
NC	60000	1	5.6E-03	1.3E-05	ND	ND			
SUMMARY HAZARD INDEX							0.008	0.000	0.008

TABLE O-22

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0-12 feet)  
CONSTRUCTION WORKER  
SETTLING POND 1  
BADGER ARMY AMMUNITION PLANT

SF15B-CW

09-Dec-92

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	400	mg/kg	USEPA, 1991
INGESTION RATE	IR	100%	mg/day	Assumption
INHALATION INGESTION	FI	1	mg/cm <sup>3</sup>	USEPA, 1992
SOIL ADHERENCE FACTOR	SAF	2,100	cm <sup>2</sup> /day	USEPA, 1990
SURFACE AREA EXPOSED	SA	0.00001	kg/mg	USEPA, 1991
CONVERSION FACTOR	CF	70	kg	USEPA, 1991
BODY WEIGHT	BW	20	days/year	USEPA, 1991
EXPOSURE FREQUENCY	EF	1	years	USEPA, 1991
EXPOSURE DURATION	ED	70	years	USEPA, 1991
AVERAGING TIME	AT	0.0247942005	years	USEPA, 1991
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	USEPA, 1999
<p>CANCER NONCANCER INGESTION DERMAL</p>				
<p> <math>\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}</math>  <math>\text{HAZARD QUOTIENT} = \text{INTAKE (mg/kg-day)} / \text{REFERENCE DOSE (mg/kg-day)}</math>  <math>\text{INTAKE} = (\text{INTAKE-INGESTION}) + (\text{INTAKE-DERMAL})</math>  <math>\text{INTAKE-INGESTION} = \text{CS} \times \text{IR} \times \text{FI} \times \text{CF} \times \text{EF} \times \text{ED}</math>  <math>\text{BW} \times \text{AT} = 365 \text{ days/yr}</math>  <math>\text{INTAKE-DERMAL} = \text{CS} \times \text{SA} \times \text{SAF} \times \text{RAF} \times \text{CF} \times \text{EF} \times \text{ED}</math>  <math>\text{BW} \times \text{AT} = 365 \text{ days/yr}</math> </p>				
<p>           Note:            For noncarcinogenic effects: AT = EF            365 days         </p>				
<p>           USEPA, 1991, Risk Assessment Guidelines for Superfund            USEPA, 1990, Exposure Factors Handbook            USEPA, 1991, Standard Default Exposure Factors         </p>				

TABLE O-22, continued  
 DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0-12 feet)  
 CONSTRUCTION WORKER  
 SETTLING POND 1  
 BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RPF	INTAKE INGESTION (mg/kg-dw)	DERMAL RPF	INTAKE DERMAL (mg/kg-dw)	CANCER SLOPE FACTOR (mg/kg-dw) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK DERMAL	TOTAL CANCER RISK
24DNT	172	1	9.2E-07	No Values Available for Quantitative Analysis		6.8E-01	6.3E-07		6.3E-07
24DNT	26	1	1.4E-07			6.8E-01	9.5E-08		9.5E-08
FB	180	1	9.7E-07			ND			
SUMMARY CANCER RISK							7E-07	6E+00	7E-07

TABLE O-22, continued  
DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0-12 feet)  
CONSTRUCTION WORKER  
SETTLING POND 1  
BADGER ARMY AMMUNITION PLANT

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAP	INTAKE INGESTION (mg/kg-day)	DERMAL RAP	INTAKE DERMAL (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT DERMAL	TOTAL HAZARD QUOTIENT
Pb	180	1	1.2E-03	No Values Available for Quantitative Analysis		ND	6.51E-04		6.51E-04
Sn	57	1	3.9E-04			6.0E-01	8.91E-04		8.91E-04
NET	13	1	8.9E-05			1.0E-01			
NE13	740	1	5.1E-03			ND			
SO4	2500	1	1.7E-02			ND			
24DNT	172	1	1.2E-03			ND			
24DNT	26	1	1.8E-04			ND			
DEP	1340	1	9.2E-03			8.0E+00	1.15E-03		1.15E-03
DNBP	14	1	9.6E-05			1.0E+00	9.60E-05		9.60E-05
DPA	10	1	6.9E-05			2.5E-02	2.74E-03		2.74E-03
2NNDPA	0.97	1	6.7E-06			ND			
NC	60000	1	4.1E-01			ND			
SUMMARY HAZARD INDEX							0.0055	0.0000	0.0055



TABLE O-23

INSULATION EXPOSURE TO AMBIENT AIR  
CONSTRUCTION WORKER  
SETTLING POND 1  
BADGER ARMY AMMUNITION PLANT

SF LARW 89-Dec-92

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL				0 zero - 12 feet see below
CONCENTRATION AIR PARTICULATES	CS	Medium	mg/kg	see below
CONCENTRATION AIR VOLATILES	CAp	Calculated	mg/m <sup>3</sup>	see below
VOLATILIZATION FACTOR	CV	Calculated	m <sup>3</sup> /kg	Appendix M
24 HOUR AVERAGE PM10 STANDARD	PM10	Calculated	ug/m <sup>3</sup>	USEPA, 1991b
CONVERSION FACTOR	CF	1.50	kg/kg	
INSULATION SLATE	IR	1E-09	kg/m <sup>2</sup> /hour	USEPA, 1991a
BODY WEIGHT	BW	2.5	kg	USEPA, 1999
EXPOSURE TIME	ET	70	hour/day	Assumption
EXPOSURE FREQUENCY	EF	8	day/year	Assumption
EXPOSURE DURATION	ED	20	years	Assumption
AVERAGING TIME	AT	1	years	Assumption
		70	years	USEPA, 1991a
		0.05-0.75-500	years	USEPA, 1991b
CANCER				
	AT			
NONCANCER				
	AT			
USEPA, 1995. Risk Assessment Guidelines for Superfund, Part A USEPA, 1991a. Standard Default Exposure Factors USEPA, 1991b. CPEL50693-877				
CANCER RISK = INTAKE (mg/kg-day) x CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup> - 1 HAZARD QUOTIENT = AIR CONCENTRATION (mg/m <sup>3</sup> ) / REFERENCE CONCENTRATION (mg/m <sup>3</sup> ) INTAKE = $\frac{(CAp + CAp) \times BW \times ET \times EF \times ED}{BW \times AT \times 365 \text{ day/yr}}$ AIR CONCENTRATION (mg/m <sup>3</sup> ) = CAp + CAv CAp = CS x PM10 x CF CAv = CS x UVF Note: For noncarcinogenic effects: AT = 365 days				

TABLE O-23, continued  
 INITIATION EXPOSURE TO AMBIENT AIR  
 CONSTRUCTION WORKER  
 SETTLING POND 1  
 BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VP (m³/kg)	AIR CONCENTRATION VOLATILES (mg/m³)	AIR CONCENTRATION PARTICULATES (mg/m³)	INTAKE (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day)⁻¹	CANCER RISK
2,4-DNT	172			0.000729	5.8E-09	ND	
2,6-DNT	26			0.000009	8.7E-10	ND	
7B	180			0.000027	6.0E-09	ND	
SUMMARY CANCER RISK							6E+00

TABLE O-23, continued  
 INSULATION EXPOSURE TO AMBIENT AIR  
 CONSTRUCTION WORKER  
 SETTLING POND 1  
 BADGER ARMY AMMUNITION PLANT

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VP (mmHg)	AIR CONCENTRATION VOLATILES (mg/m <sup>3</sup> )	AIR CONCENTRATION PARTICULATES (mg/m <sup>3</sup> )	REFERENCE CONCENTRATION (mg/m <sup>3</sup> )	HAZARD QUOTIENT VOLATILES	HAZARD QUOTIENT PARTICULATES	HAZARD QUOTIENT TOTAL
FB	180			0.000027	ND			
SN	57			0.00000835	ND			
NET	13			0.00000195	ND			
NE3	740			0.00011	ND			
SN4	2500			0.000375	ND			
3CHNT	172			0.000258	ND			
2CHNT	26			0.000039	ND			
DHP	460			0.000069	ND			
DHP2	14			0.000021	ND			
DFA	10			0.000015	ND			
2CHDFA	0.97			0.000001435	ND			
NC	6000			0.009	ND			
SUMMARY HAZARD INDEX								
							0	0

**Table O-24**  
**Compounds Detected**  
**Settling Pond 2**  
**Settling Ponds and Spoils Disposal Area Surface Soil (0-2')**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment (Y/N)?</u>	<u>Reason *</u>	<u>Exposure Point Concentration **</u>
AL	3 : 3	40000	12000	N	1	
PB	3 : 3	250	95	Y		250
K	3 : 3	600	370	N	1, 3	
NA	3 : 3	120	72	N	1, 3	
SN	3 : 3	53	22	Y		53
NIT	3 : 3	43	14	Y		43
NH3	3 : 3	840	260	Y		840
SO4	1 : 3	64		Y		64
24DNT	1 : 4	7.6		Y		7.6
DEP	1 : 4	135		Y		135
DNBP	1 : 4	0.74		Y		0.74
DPA	1 : 3	1.5		Y		1.5
NC	2 : 3	280	260	Y		280

Footnotes:

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential for human nutrition.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum

Note: Assessment of surface soil contamination (0 to 2 feet) was performed using data from samples FPII-1 through FPII-3 and S1205.

Table O-25  
Compounds Detected  
Settling Pond 2  
Settling Ponds and Spoils Disposal Area Subsurface Soil (2'-16')  
Units: ug/g

Remedial Investigation  
Badger Army Ammunition Plant

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	<u>Concentration **</u>
24DNT	1 : 1	0.04	-	Y		0.04
AL	1 : 1	3750	-	Y		3750
PB	1 : 1	30	-	N	1	
SN	1 : 1	4.7	-	Y		4.7
SO4	1 : 1	20.2	-	Y		20.2

Footnotes:

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential for human nutrition.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum.

Note: Assessment of subsurface soil contamination (2 to 16 feet) was performed using data from sample S1205.

TABLE O-26

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SETTLING POND 2  
BADGER ARMY AMMUNITION PLANT

SF 28330E

25 - Mar - 93

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Medium	mg/kg	USEPA 1991
INGESTION RATE - ADULT	IRa	100	mg/day	USEPA 1991
INGESTION RATE - CHILD	IRc	200	mg/day	USEPA 1991
FRACTION INGESTED	FI	100%		Assumption
CONVERSION FACTOR	CF	0.000001	kg/mg	USEPA 1991
BODY WEIGHT - ADULT	BWa	70	kg	USEPA 1991
BODY WEIGHT - CHILD	BWc	15	kg	USEPA 1991
EXPOSURE FREQUENCY	EF	350	days/year	USEPA 1991
EXPOSURE DURATION - ADULT	EDa	24	years	USEPA 1991
EXPOSURE DURATION - CHILD	EDc	6	years	USEPA 1991
AVERAGING TIME	AT	70	years	USEPA 1991
CANCER	ATa	24	years	USEPA 1991
ADULT - NONCANCER	ATc	6	years	USEPA 1991
CHILD - NONCANCER	RAF	1	unitless	USEPA 1991
RELATIVE ABSORPTION FACTOR				
Note: For noncarcinogenic effects: AT = ED				
CANCER RISK = INTAKE (mg/kg-day) x CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup> HAZARD QUOTIENT = INTAKE (mg/kg-day) / REFERENCE DOSE (mg/kg-day) INTAKE-ADULT = $\frac{CS \times IRa \times FI \times CF \times EF \times EDa}{BWa \times ATa \times 365 \text{ days/yr}}$ INTAKE-CHILD = $\frac{CS \times IRc \times FI \times CF \times EF \times EDc}{BWc \times ATc \times 365 \text{ days/yr}}$				

TABLE O-2A, continued  
INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SETTLING POND 2  
BADGER ARMY AMMUNITION PLANT

872835W8 25-Mar-92

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RPF	INTAKE		CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK ADULT	CANCER RISK CHILD	TOTAL CANCER RISK
			ADULT (mg/kg-day)	CHILD (mg/kg-day)				
AGENT PB	7.6	1	3.6E-06	8.3E-06	6.8E-01	2.4E-06	5.7E-06	8.1E-06
	250	1	1.2E-04	2.7E-04	ND			
SUMMARY CANCER RISK						2E-06	6E-06	8E-06

TABLE O-24, continued  
INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SETTLING POND 2  
BADGER ARMY AMMUNITION PLANT

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RPF	INTAKE ADULT (mg/kg-day)	INTAKE CHILD (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT ADULT	HAZARD QUOTIENT CHILD	TOTAL HAZARD QUOTIENT
PB	230	1	3.4E-04	3.2E-03	ND	1.21E-04	1.13E-03	1.25E-03
SN	53	1	7.3E-05	6.8E-04	6.0E-01	5.89E-04	5.50E-03	6.09E-03
MT	43	1	5.9E-05	5.5E-04	1.0E-01			
HEX	840	1	1.2E-03	1.1E-02	ND			
SO4	64	1	8.8E-05	8.2E-04	ND			
300NT	7.6	1	1.0E-05	9.7E-05	2.0E-03	5.21E-05	4.86E-02	5.38E-02
DNP	135	1	1.8E-04	1.7E-03	8.0E-01	2.51E-04	2.16E-03	2.39E-03
DNEP	0.74	1	1.0E-06	9.5E-06	1.0E-01	1.01E-05	9.46E-03	1.05E-04
DPA	1.5	1	2.1E-06	1.9E-05	2.5E-02	8.22E-05	7.67E-04	8.49E-04
NC	280	1	3.8E-04	3.6E-03	ND			
SUMMARY HAZARD INDEX						0.006	0.033	0.034



**TABLE O-27**  
**INCIDENTAL INGESTION AND INHALATION OF SOIL**  
**GROUNDNS MAINTENANCE WORKER**  
**SETTLING POND 2**  
**BADGER ARMY AMMUNITION PLANT**

## EXPOSURE PARAMETERS

## EQUATIONS

[illegible]

TABLE O-27, continued  
 INCIDENTAL INGESTION AND INHALATION OF SOIL  
 GROUNDS MAINTENANCE WORKER  
 SETTILING POND 2  
 BADGER ARMY AMMUNITION PLANT

## CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION EAF	ESTAKE INGESTION (mg/kg-day)	INTAKE INHALATION (mg/kg-day)	CANCER SLOPE FACTOR-DNL (mg/kg-day) <sup>-1</sup>	CANCER SLOPE FACTOR-DNL (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK INHALATION	TOTAL CANCER RISK
24DNT PB	7.6 250	1 1	2.5E-07 8.4E-06	1.1E-11 3.6E-10	ND ND	6.8E-01	1.7E-07		1.7E-07
SUMMARY CANCER RISK							2E-07	6E+00	2E-07

TABLE O-27, continued  
INCIDENTAL INGESTION AND INHALATION OF SOIL  
GROUNDS MAINTENANCE WORKER  
SETTLING POND 2  
BADGER ARMY AMMUNITION PLANT

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION BAF	INTAKE INGESTION (mg/kg-day)	AIR CONCENT. (mg/m <sup>3</sup> )	REFERENCE CONCENT. (mg/m <sup>3</sup> )	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT INHALATION	TOTAL HAZARD QUOTIENT
PB	250	1	2.3E-05	5.4E-08	ND	ND	8.30E-06		8.30E-06
SN	53	1	5.0E-06	1.1E-08	ND	6.0E-01	4.04E-05		4.04E-05
NET	43	1	4.0E-06	9.3E-09	ND	1.0E-01			
NH3	840	1	7.9E-05	1.8E-07	ND	ND			
BO4	64	1	6.0E-06	1.4E-08	ND	ND			
24DNT	7.6	1	7.1E-07	1.6E-09	ND	2.0E-03	3.57E-04		3.57E-04
DEP	135	1	1.3E-05	2.9E-08	ND	8.0E-01	1.99E-05		1.99E-05
DNBP	0.74	1	7.0E-08	1.6E-10	ND	1.0E-01	6.95E-07		6.95E-07
DPA	1.5	1	1.4E-07	3.3E-10	ND	2.0E-02	7.05E-06		7.05E-06
NC	280	1	2.6E-05	6.0E-08	ND	ND			
SUMMARY HAZARD INDEX							0.0004	0.0008	0.0004

**TABLE 0-28**  
**DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0 - 12 feet)**  
**CONSTRUCTION WORKER**  
**SETTLING POND 2**  
**BADGER ARMY AMMUNITION PLANT**

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Medium	mg/kg	USEPA, 1991
INGESTION RATE	IR	400	mg/day	Assumption
FRACTION INGESTED	FI	100%		
SOIL ADHERENCE FACTOR	SAF	1	mg/cm <sup>2</sup>	USEPA, 1992
SURFACE AREA EXPOSED	SA	2,100	cm <sup>2</sup> /day	USEPA, 1990
CONVERSION FACTOR	CF	0.000001	kg/mg	
BODY WEIGHT	BW	70	kg	USEPA, 1991
EXPOSURE FREQUENCY	EF	20	days/year	USEPA, 1991
EXPOSURE DURATION	ED	1	years	USEPA, 1991
AVERAGING TIME				
CANCER	AT	70	years	USEPA, 1999
NONCANCER	AT	0.0547943205	years	USEPA, 1991
RELATIVE ABSORPTION FACTOR	RAF		unitless	USEPA, 1999
INGESTION				
DERMAL				

CANCER RISK = INTAKE (mg/kg-day) x CANCER SLOPE FACTOR (mg/kg-day)<sup>-1</sup>

HAZARD QUOTIENT = INTAKE (mg/kg-day) / REFERENCE DOSE (mg/kg-day)

INTAKE = (INTAKE-INGESTION) + (INTAKE-DERMAL)

INTAKE-INGESTION =  $\frac{CS \times IR \times RAF \times FI \times CF \times EF \times ED}{BW \times AT \times 365 \text{ days/yr}}$

INTAKE-DERMAL =  $\frac{CS \times SA \times SAF \times RAF \times CF \times EF \times ED}{BW \times AT \times 365 \text{ days/yr}}$

Note:

For noncardiotoxic effects: AT = EF

365 days

USEPA, 1999. Risk Assessment Guidelines for Superfund

USEPA, 1990. Exposure Factors Handbook

USEPA, 1991. Standard Default Exposure Factors

USEPA, 1992. Dermal Exposure Guidelines

TABLE O-2A, continued  
DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0 - 12 feet)  
CONSTRUCTION WORKER  
SETTLING POND 2  
BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAI	INTAKE INGESTION (mg/kg-day)	DERMAL RAI	INTAKE DERMAL (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK DERMAL	TOTAL CANCER RISK
2,4-DNT PB	7.6 250	1 1	4.1E-06 1.3E-06	No Values for Quantitative Analysis		6.8E-01 ND	2.8E-06		2.8E-06
SUMMARY CANCER RISK							3E-06	6E-06	3E-06

TABLE O-2A, continued  
DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0 - 12 feet)  
CONSTRUCTION WORKER  
SETTLING POND 2  
BADGER ARMY AMMUNITION PLANT

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAP	INTAKE INGESTION (mg/kg-day)	DERMAL RAP	INTAKE DERMAL (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT DERMAL	TOTAL HAZARD QUOTIENT
Pb	250	1	1.7E-03	No Values Available for Quantitative Analysis		ND	6.06E-04		6.06E-04
SN	53	1	3.6E-04			6.0E-01	2.95E-03		2.95E-03
NET	43	1	2.9E-04			1.0E-01			
NI13	840	1	5.8E-03			ND			
SO4	64	1	4.4E-04			ND			
24DNT	7.6	1	5.2E-05			ND			
DEP	135	1	9.3E-04			8.0E+00	1.16E-04		1.16E-04
DINBP	0.74	1	5.1E-06			1.0E+00	5.07E-06		5.07E-06
DPA	1.5	1	1.0E-05			2.5E-02	4.11E-04		4.11E-04
NC	280	1	1.9E-03			ND			
SUMMARY HAZARD INDEX							0.0041	0.0006	0.0041

TABLE O-29

INITIALATION EXPOSURE TO AMBIENT AIR  
CONSTRUCTION WORKER  
SETTLING POND 2  
BADGER ARMY AMMUNITION PLANT

EF2ARW 89-Dec-92

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Maximum	mg/kg	0 zero - 12 feet
CONCENTRATION AIR PARTICULATES	CAp	Calculated	mg/m <sup>3</sup>	see below
CONCENTRATION AIR VOLATILES	CAv	Calculated	mg/m <sup>3</sup>	see below
VOLATILIZATION FACTOR	VF	Calculated	m <sup>3</sup> /kg	Appendix M
24 HOUR AVERAGE PM10 STANDARD	PM10	150	ug/m <sup>3</sup>	USEPA, 1991b
CONVERSION FACTOR	CF	1E-09	kg/mg	
INHALATION RATE	IhR	2.5	m <sup>3</sup> /hour	USEPA, 1991a
BODY WEIGHT	BW	70	kg	USEPA, 1989
EXPOSURE TIME	ET	8	hour/day	Assumption
EXPOSURE FREQUENCY	EF	20	day/year	Assumption
EXPOSURE DURATION	ED	1	years	Assumption
AVERAGING TIME	AT	70	years	USEPA, 1991a
CANCER	AT	0.05-0.945365	years	USEPA, 1991a
NONCANCER	AT			USEPA, 1991a
USEPA, 1989, Risk Assessment Guidelines for Superfund, Part A				
USEPA, 1991a, Standard Default Exposure Factors				
USEPA, 1991b, CFR 50.693-697				

$$\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$$

$$\text{HAZARD QUOTIENT} = \text{AIR CONCENTRATION (mg/m}^3\text{)} / \text{REFERENCE CONCENTRATION (mg/m}^3\text{)}$$

$$\text{INTAKE} = \frac{(CAp + CAv) \times IhR \times ET \times EF \times ED}{BW \times AT} \times 365 \text{ days/yr}$$

$$\text{AIR CONCENTRATION (mg/m}^3\text{)} = CAp + CAv$$

$$CAp = CS \times PM10 \times CF$$

$$CAv = CS \times VF$$

Note:

For noncarcinogenic effects: AT = EF  
365 days

TABLE O-29, continued  
 INITIATION EXPOSURE TO AMBIENT AIR  
 CONSTRUCTION WORKER  
 SETTLING POND 2  
 BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VP (m <sup>3</sup> /kg)	AIR CONCENTRATION VOLATILES (mg/m <sup>3</sup> )	AIR CONCENTRATION PARTICULATES (mg/m <sup>3</sup> )	INTAKE (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK
ACENT 78	7.6 250			0.00000114 0.0000375	2.5E-10 8.4E-09	ND ND	
SUMMARY CANCER RISK							0E+00



TABLE O-29, continued  
 INITIALATION EXPOSURE TO AMBIENT AIR  
 CONSTRUCTION WORKER  
 SETTLING POND 2  
 BADGER ARMY AMMUNITION PLANT

892AEW

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VF (m <sup>3</sup> /kg)	AIR CONCENTRATION VOLATILES (mg/m <sup>3</sup> )	AIR CONCENTRATION PARTICULATES (mg/m <sup>3</sup> )	REFERENCE CONCENTRATION (mg/m <sup>3</sup> )	HAZARD QUOTIENT VOLATILES	HAZARD QUOTIENT PARTICULATES	HAZARD QUOTIENT TOTAL
FB	250			0.0000375	ND			
SN	55			0.0000795	ND			
MTT	45			0.00000645	ND			
NEL3	840			0.000126	ND			
S04	64			0.0000096	ND			
3MDHT	7.6			0.00000114	ND			
DEP	135			0.00002025	ND			
DNEP	0.74			0.000000111	ND			
DPA	1.5			0.000000225	ND			
INC	280			0.000042	ND			
SUMMARY HAZARD INDEX								0

**Table O-30**  
**Compounds Detected**  
**Settling Pond 3**  
**Settling Ponds and Spoils Disposal Area Surface Soil (0-2')**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
AL	15 : 15	34000	2900	N	1	
PB	15 : 15	34	6.7	N	1	
K	15 : 15	1300	140	N	1, 3	
NA	15 : 15	160	1.1	N	1, 3	
SN	15 : 15	72	23	Y		72
NIT	15 : 15	4.9	0.39	Y		4.9
NH3	15 : 15	520	21	Y		520
SO4	2 : 15	36	30	Y		36
24DNT	1 : 16	2.6		Y		2.6
26DNT	1 : 15	1.5		Y		1.5
DEP	1 : 16	44		Y		44
DNBP	5 : 16	17.4	2.5	Y		17.4
DPA	4 : 15	2.8	0.24	Y		2.8
NC	2 : 15	190	50	Y		190

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant.  
                   \* 3 = essential for human nutrition.  
                   \* 4 = frequency of detection less than 5 %.  
                   \*\* 95th percentile or maximum

Note:           Assessment of surface soil contamination (0 to 2 feet) was performed  
                   using data from samples FP111-1 through FP111-15 and S1206.

Table O-31  
Compounds Detected  
Settling Pond 3  
Settling Ponds and Spoils Disposal Area Subsurface Soil (2'-16')  
Units: ng/g

Remedial Investigation  
Badger Army Ammunition Plant

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	<u>Concentration **</u>
24DNT	1 : 1	0.057	-	Y		0.057
AL	1 : 1	1750	-	N	1	
NC	1 : 1	0.17	-	Y		0.17
PB	1 : 1	20	-	N	1	
SN	1 : 1	3.9	-	Y		3.9
SO4	1 : 1	15.2	-	Y		15.2

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant.  
                   \* 3 = essential for human nutrition.  
                   \* 4 = frequency of detection less than 5 %.  
                   \*\* 95th percentile or maximum.

Note:           Assessment of subsurface soil contamination (2 to 16 feet) was performed  
                   using data from boring S1206.

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SETTLING POND 3  
BADGER ARMY AMMUNITION PLANT

SP-35550E

25-Mar-93

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Medium	mg/kg	USEPA, 1991
INGESTION RATE - ADULT	IRa	100	mg/day	USEPA, 1991
INGESTION RATE - CHILD	IRc	200	mg/day	USEPA, 1991
FRACTION INGESTED	FI	100%		Assumption
CONVERSION FACTOR	CF	0.000001	kg/kg	USEPA, 1991
BODY WEIGHT - ADULT	BWa	70	kg	USEPA, 1991
BODY WEIGHT - CHILD	BWc	15	kg	USEPA, 1991
EXPOSURE FREQUENCY	EF	350	days/year	USEPA, 1991
EXPOSURE DURATION - ADULT	EDa	34	years	USEPA, 1991
EXPOSURE DURATION - CHILD	EDc	6	years	USEPA, 1991
AVERAGING TIME	AT	70	years	USEPA, 1989
CANCER	ATc	24	years	USEPA, 1991
ADULT - NONCANCER	ATc	6	years	USEPA, 1991
CHILD - NONCANCER	RAF	1	unitless	USEPA, 1989
RELATIVE ABSORPTION FACTOR				

<p>CANCER RISK = INTAKE (mg/kg-day) x CANCER SLOPE FACTOR (mg/kg-day)<sup>-1</sup></p> <p>HAZARD QUOTIENT = INTAKE (mg/kg-day) / REFERENCE DOSE (mg/kg-day)</p> <p>INTAKE-ADULT = <math>\frac{CS \times IRa \times RAF \times FI \times CF \times EF \times EDa}{BWa \times ATa \times 365 \text{ days/yr}}</math></p> <p>INTAKE-CHILD = <math>\frac{CS \times IRc \times RAF \times FI \times CF \times EF \times EDc}{BWc \times ATc \times 365 \text{ days/yr}}</math></p>	<p>Note:</p> <p>For noncarcinogenic effects: AT = ED</p>
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USEPA, 1989. Risk Assessment Guidelines for Superfund  
USEPA, 1991. Standard Default Exposure Factors

TABLE O-32, continued  
INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SETTLING POND 3  
BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAF	INTAKE ADULT (mg/kg-day)	INTAKE CHILD (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK ADULT	CANCER RISK CHILD	TOTAL CANCER RISK
2,4-DNT	2.6	1	1.2E-06	2.8E-06	6.8E-01	8.3E-07	1.9E-06	2.8E-06
2,6-DNT	1.5	1	7.0E-07	1.6E-06	6.8E-01	4.8E-07	1.1E-06	1.6E-06
FB	34	1	1.6E-05	3.7E-05	ND			
SUMMARY CANCER RISK								
						1E-06	3E-06	4E-06

TABLE O-32, continued  
INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL -- ADULT AND CHILD  
SETTLING POND 3  
BADGER ARMY AMMUNITION PLANT

SP-56306 25-Mar-93

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION LAF	INTAKE		REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT		HAZARD QUOTIENT	
			ADULT (mg/kg-day)	CHILD (mg/kg-day)		ADULT	CHILD	ADULT	CHILD
PB	34	1	4.7E-03	4.3E-04	ND				
SN	72	1	9.9E-03	9.2E-04	6.0E-01	1.64E-04	1.53E-03	1.70E-03	1.70E-03
MTT	4.9	1	6.7E-06	6.2E-03	1.0E-01	6.71E-05	6.26E-04	6.94E-04	6.94E-04
MEB	570	1	7.1E-04	6.6E-03	ND				
SO4	36	1	4.9E-03	4.6E-04	ND				
2,4-DNT	2.6	1	3.6E-06	3.3E-03	2.0E-03	1.78E-10	1.66E-02	1.84E-02	1.84E-02
2,6-DNT	1.5	1	2.1E-06	1.9E-03	ND				
DIB	44	1	6.0E-03	5.6E-04	8.0E-01	7.53E-05	7.01E-04	7.79E-04	7.79E-04
DNEP	17.4	1	2.4E-03	2.2E-04	1.0E-01	2.38E-04	2.22E-03	2.46E-03	2.46E-03
DFA	2.8	1	3.6E-06	3.6E-03	2.5E-02	1.53E-04	1.43E-03	1.59E-03	1.59E-03
NC	190	1	2.6E-04	2.4E-03	ND				
SUMMARY HAZARD INDEX						0.0023	0.0231	0.0236	0.0236

TABLE O-33

INCIDENTAL INGESTION AND INHALATION OF SOIL  
 GROUNDS MAINTENANCE WORKER  
 SETTLING POND 3  
 BADGER ARMY AMMUNITION PLANT

SPASS(TW) 08-Dec-92

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Maximum	mg/kg	USEPA, 1991a Assumption
INGESTION RATE	IR	100	mg/day	
FRACTION INGESTED	FI	100%		
CONVERSION FACTOR	CF	0.000001	kg/mg	
BODY WEIGHT	BW	70	kg	USEPA, 1991a
EXPOSURE FREQUENCY	EF	24	days/year	USEPA, 1991a
EXPOSURE DURATION	ED	25	years	USEPA, 1991a
CONCENTRATION AIR PARTICULATES	CAp	Calculated	mg/m <sup>3</sup>	Appendix M
CONCENTRATION AIR VOLATILES	CAv	Calculated	mg/m <sup>3</sup>	USEPA, 1991b
VOLATILIZATION FACTOR	VF	Calculated	m <sup>3</sup> /kg	USEPA, 1991a
PARTICULATE EMISSION FACTOR	PEF	4.63E+09	m <sup>3</sup> /kg	Assumption
INHALATION RATE	IhR	2.5	m <sup>3</sup> /hour	
EXPOSURE TIME	ET	8	hours/day	
AVERAGING TIME				
CANCER	AT	70	years	USEPA, 1999
NONCANCER	AT	25	years	USEPA, 1991a
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	USEPA, 1999

USEPA, 1999. Risk Assessment Guidelines for Superfund - Part A

USEPA, 1990. Exposure Factors Handbook

USEPA, 1991a. Standard Default Exposure Factors

USEPA, 1991b. Risk Assessment Guidelines for Superfund - Part B

$$\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$$

$$\text{HAZARD QUOTIENT}_{\text{Ingestion}} = \text{INTAKE (mg/kg-day)} / \text{REFERENCE DOSE (mg/kg-day)}$$

$$\text{HAZARD QUOTIENT}_{\text{Inhalation}} = \frac{\text{AIR CONCENTRATION (mg/m}^3\text{)}}{\text{REFERENCE CONCENTRATION (mg/m}^3\text{)}}$$

$$\text{INTAKE - INGESTION} = \frac{\text{CS} \times \text{IR} \times \text{RAF} \times \text{FI} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

$$\text{INTAKE - INHALATION} = \frac{(\text{CAp} + \text{CAv}) \times \text{IhR} \times \text{ET} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

$$\text{AIR CONCENTRATION (mg/m}^3\text{)} = \text{CAp} + \text{CAv}$$

$$\text{CAp} = \text{CS} \times \text{VF} \times \text{PEF}$$

$$\text{CAv} = \text{CS} \times \text{VVF}$$

Note:

For noncarcinogenic effects: AT = ED

TABLE O-31, continued

INCIDENTAL INGESTION AND INHALATION OF SOIL  
 GROUNDS MAINTENANCE WORKER  
 SETTLING POND 3  
 BADGER ARMY AMMUNITION PLANT

SP3SSOW 08-Dec-92

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RPF	INTAKE INGESTION (mg/kg-day)	INTAKE INHALATION (mg/kg-day)	CANCER SLOPE FACTOR-INEL (mg/kg-day) <sup>-1</sup>	CANCER SLOPE FACTOR-ING. (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK INHALATION	TOTAL CANCER RISK
24DNT	2.6	1	8.7E-08	3.8E-12	ND	6.8E-01	5.9E-08		5.9E-08
24DNT	1.5	1	5.0E-08	2.2E-12	ND	6.8E-01	3.4E-08		3.4E-08
PB	34	1	1.1E-06	4.9E-11	ND				
SUMMARY CANCER RISK									9E-08
									0E+00



TABLE O-33, continued

INCIDENTAL INGESTION AND INHALATION OF SOIL  
 GROUNDS MAINTENANCE WORKER  
 SETTLING POND 3  
 BADGER ARMY AMMUNITION PLANT

SPSSG/W 08-Dec-92

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION EAF	INGESTION (mg/kg-day)	AIR CONCENT. (mg/m <sup>3</sup> )	REFERENCE CONCENT. (mg/m <sup>3</sup> )	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT INHALATION	TOTAL HAZARD QUOTIENT
PB	34	1	3.2E-06	7.3E-09	ND	ND	1.13E-05		1.13E-05
SN	72	1	6.8E-06	1.6E-08	ND	6.0E-01	1.13E-05		1.13E-05
NTT	4.9	1	4.6E-07	1.1E-09	ND	1.0E-01	4.60E-06		4.60E-06
NEIS	520	1	4.9E-05	1.1E-07	ND	ND			
SO4	36	1	3.4E-06	7.8E-09	ND	ND			
24DNT	2.6	1	2.4E-07	5.6E-10	ND	2.0E-03	1.22E-04		1.22E-04
26DNT	1.5	1	1.4E-07	3.2E-10	ND	ND			
DEP	44	1	4.1E-06	9.5E-09	ND	8.0E-01	5.17E-06		5.17E-06
DNEP	17.4	1	1.6E-06	3.8E-09	ND	1.0E-01	1.63E-05		1.63E-05
DPA	2.8	1	2.6E-07	6.0E-10	ND	2.0E-02	1.32E-05		1.32E-05
NC	190	1	1.8E-05	4.1E-08	ND	ND			
SUMMARY HAZARD INDEX							0.0002	0.0000	0.0002

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0-12 feet)  
CONSTRUCTION WORKER  
SETTLING POND 3  
BADGER ARMY AMMUNITION PLANT

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	C3	Median	mg/kg	USEPA, 1991
INGESTION RATE	IR	480	mg/day	Assumption
FRACTION INGESTED	FI	100%		USEPA, 1992
SOIL ADHERENCE FACTOR	SAF	1	mg/cm <sup>2</sup>	USEPA, 1990
SURFACE AREA EXPOSED	SA	2,100	cm <sup>2</sup> /day	USEPA, 1991
CONVERSION FACTOR	CF	0.000001	kg/mg	USEPA, 1991
BODY WEIGHT	BW	70	kg	USEPA, 1991
EXPOSURE FREQUENCY	EF	20	days/year	USEPA, 1991
EXPOSURE DURATION	ED	1	years	USEPA, 1991
AVERAGING TIME	AT	70	years	USEPA, 1989
RELATIVE ABSORPTION FACTOR	RAF	0.0547945203	years	USEPA, 1991
		1	unitless	USEPA, 1989
<p> CANCER  NONCANCER  INGESTION  DERMAL </p>				
<p> CANCER RISK = <math>\text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}</math>  HAZARD QUOTIENT = <math>\text{INTAKE (mg/kg-day)} / \text{REFERENCE DOSE (mg/kg-day)}</math>  INTAKE = <math>(\text{INTAKE-INGESTION}) + (\text{INTAKE-DERMAL})</math>  INTAKE-INGESTION = <math>\frac{\text{C3} \times \text{IR} \times \text{SAF} \times \text{FI} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}</math>  INTAKE-DERMAL = <math>\frac{\text{C3} \times \text{SA} \times \text{SAF} \times \text{RAF} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}</math> </p>				
<p> Note:  For noncarcinogenic effects: AT = EF  365 days </p>				
<p> USEPA, 1989, Risk Assessment Guidelines for Superfund  USEPA, 1990, Exposure Factors Handbook  USEPA, 1991, Standard Default Exposure Factors </p>				
USEPA, 1992, Dermal Exposure Guidelines				

TABLE O-34, continued

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0-12 feet)

CONSTRUCTION WORKER

SETTLING POND 3

BADGER ARMY AMMUNITION PLANT

## CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RPF	INTAKE INGESTION (mg/kg-day)	DERMAL RPF	INTAKE DERMAL (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK DERMAL	TOTAL CANCER RISK
24DNT	2.6	1	1.4E-08	No Values		6.8E-01	9.5E-09		9.5E-09
26DNT	1.5	1	8.1E-09	Available		6.8E-01	5.5E-09		5.5E-09
PB	34	1	1.8E-07	for Quantitative Analysis		ND			
SUMMARY CANCER RISK							1E-08	6E+00	1E-08

## DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0-12 feet)

CONSTRUCTION WORKER

SETTLING POND 3

BADGER ARMY AMMUNITION PLANT

## NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAP	INGESTION (mg/kg-day)	DERMAL RAP	DERMAL (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT DERMAL	TOTAL HAZARD QUOTIENT
PB	34	1	2.3E-04	No Values		ND			
SN	72	1	4.9E-04	Available		6.0E-01	8.23E-04		8.23E-04
NTT	4.9	1	3.4E-05	for		1.0E-01	3.36E-04		3.36E-04
NI13	520	1	3.6E-03	Quantitative		ND			
SO4	36	1	2.5E-04	Analysis		ND			
24DNT	2.6	1	1.8E-05			ND			
24DNT	1.5	1	1.0E-05			ND			
DEP	44	1	3.0E-04			8.0E+00	3.77E-05		3.77E-05
DNBP	17.4	1	1.2E-04			1.0E+00	1.19E-04		1.19E-04
DPA	2.8	1	1.9E-05			2.5E-02	7.68E-04		7.68E-04
NC	190	1	1.3E-03			ND			
SUMMARY HAZARD INDEX							0.0021	0.0000	0.0021

TABLE O-35

INITIATION EXPOSURE TO AMBIENT AIR  
CONSTRUCTION WORKER  
SETTLING POND 3  
BADGER ARMY AMMUNITION PLANT

SFJRW 09-Dec-92

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Medium	mg/kg	@ zero - 12 feet
CONCENTRATION AIR PARTICULATES	CAp	Calculated	mg/m <sup>3</sup>	see below
CONCENTRATION AIR VOLATILES	CAV	Calculated	mg/m <sup>3</sup>	see below
VOLATILIZATION FACTOR	VF	Calculated	ug/m <sup>3</sup>	Appendix M
24 HOUR AVERAGE PM10 STANDARD	PM10	150	ug/m <sup>3</sup>	USEPA, 1991b
CONVERSION FACTOR	CF	1E-09	kg/ug	USEPA, 1991b
INHALATION RATE	IR	2.5	m <sup>3</sup> /hour	USEPA, 1991a
BODY WEIGHT	BW	70	kg	USEPA, 1989
EXPOSURE TIME	ET	8	hours/day	Assumption
EXPOSURE FREQUENCY	EF	20	days/year	Assumption
EXPOSURE DURATION	ED	1	years	Assumption
AVERAGING TIME	AT	70	years	USEPA, 1991a
CANCER	AT	0.0547945203	years	USEPA, 1991a
NONCANCER	AT			
USEPA, 1989. Risk Assessment Guidance for Superfund, Part A				
USEPA, 1991a. Standard Default Exposure Factors				
USEPA, 1991b. CFR 50695 - 07				

$$\text{CANCER RISK} = \text{INTAKE} (\text{mg/kg-day}) \times \text{CANCER SLOPE FACTOR} (\text{mg/kg-day})^{-1}$$

$$\text{HAZARD QUOTIENT} = \text{AIR CONCENTRATION} (\text{mg/m}^3) / \text{REFERENCE CONCENTRATION} (\text{mg/m}^3)$$

$$\text{INTAKE} = \frac{(\text{CAp} + \text{CAV}) \times \text{IR} \times \text{ET} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

$$\text{AIR CONCENTRATION} (\text{mg/m}^3) = \text{CAp} + \text{CAV}$$

$$\text{CAp} = \text{CS} \times \text{PM10} \times \text{CF}$$

$$\text{CAV} = \text{CS} \times \text{VF}$$

Note:

For noncardiogenic effects: AT = EF

365 days

TABLE O-35, continued  
INITIALATION EXPOSURE TO AMBIENT AIR  
CONSTRUCTION WORKER  
SETTLING POND 3  
BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VP ( $\text{m}^3/\text{kg}$ )	AIR CONCENTRATION VOLATILES ( $\text{mg}/\text{m}^3$ )	AIR CONCENTRATION PARTICULATES ( $\text{mg}/\text{m}^3$ )	INTAKE ( $\text{mg}/\text{kg}-\text{day}$ )	CANCER SLOPE FACTOR ( $\text{mg}/\text{kg}-\text{day}$ ) <sup>-1</sup>	CANCER RISK
3,4-DNT	2.6			0.0000039	8.7E-11	ND	
2,6-DNT	1.5			0.0000023	5.0E-11	ND	
FB	34			0.0000051	1.1E-09	ND	
SUMMARY CANCER RISK							0E+00

TABLE O-35, continued  
 INITIALATION EXPOSURE TO AMBIENT AIR  
 CONSTRUCTION WORKER  
 SETTLING POND 3  
 BADGER ARMY AMMUNITION PLANT

SPJALW

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VF ( $\mu^2/\text{kg}$ )	AIR CONCENTRATION VOLATILES (mg/m <sup>3</sup> )	AIR CONCENTRATION PARTICULATES (mg/m <sup>3</sup> )	REFERENCE CONCENTRATION (mg/kg)	HAZARD QUOTIENT VOLATILES	HAZARD QUOTIENT PARTICULATES	HAZARD QUOTIENT TOTAL
FB	34			0.0000051	ND			
EN	72			0.0000106	ND			
NTT	4.9			0.00000735	ND			
NEB	520			0.000078	ND			
SO	36			0.0000054	ND			
2ADNT	2.6			0.00000039	ND			
2EDNT	1.5			0.000000235	ND			
DIB	44			0.0000066	ND			
DNEP	17.4			0.0000034	ND			
DPA	2.8			0.00000042	ND			
NC	190			0.0000285	ND			
SUMMARY HAZARD INDEX								
						0	0	0

**Table O-36**  
**Compounds Detected**  
**Settling Pond 4**  
**Settling Ponds and Spoils Disposal Area Surface Soil (0-2')**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	Retained for Risk Assessment		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
AL	11 : 11	60000	1300	Y		60000
PB	11 : 11	300	8.4	Y		300
K	10 : 10	1900	25	N	1, 3	
NA	9 : 10	400	44	N	1, 3	
SN	11 : 11	77	1.1	Y		77
NIT	10 : 11	10	0.67	Y		10
NH3	10 : 10	960	29	Y		960
SO4	3 : 11	400	170	Y		400
DPA	1 : 10	0.36		Y		0.36
NC	2 : 11	1038	50	Y		1038

Footnotes:

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential for human nutrition.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum

Note: Assessment of surface soil contamination (0 to 2 feet) was performed using samples FPIV-1 through FPIV-10 and S1207.



TABLE O-37

INCIDENTAL INGESTION OF SURFACE SOIL.  
RESIDENTIAL - ADULT AND CHILD  
SETTLING POND 4  
BADGER ARMY AMMUNITION PLANT

SP-613508 25-Mar-93

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Minimum	mg/kg	USEPA, 1991
INGESTION RATE - ADULT	IRa	100	mg/day	USEPA, 1991
INGESTION RATE - CHILD	IRc	200	mg/day	USEPA, 1991
FRACTION INGESTED	FI	100%		Assumption
CONVERSION FACTOR	CF	0.000001		
BODY WEIGHT - ADULT	BW <sub>a</sub>	70	kg	USEPA, 1991
BODY WEIGHT - CHILD	BW <sub>c</sub>	15	kg	USEPA, 1991
EXPOSURE FREQUENCY	EF	350	days/year	USEPA, 1991
EXPOSURE DURATION - ADULT	ED <sub>a</sub>	24	years	USEPA, 1991
EXPOSURE DURATION - CHILD	ED <sub>c</sub>	6	years	USEPA, 1991
AVERAGING TIME				
CANCER	AT	70	years	USEPA, 1989
ADULT - NONCANCER	AT <sub>a</sub>	24	years	USEPA, 1991
CHILD - NONCANCER	AT <sub>c</sub>	6	years	USEPA, 1991
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	USEPA, 1989

USEPA, 1989. Risk Assessment Guidelines for Superfund  
USEPA, 1991. Standard Default Exposure Factors

Note:  
For noncarcinogenic effects: AT = ED

$$\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$$

$$\text{HAZARD QUOTIENT} = \text{INTAKE (mg/kg-day)} / \text{REFERENCE DOSE (mg/kg-day)}$$

$$\text{INTAKE-ADULT} = \frac{CS \times IRa \times RAF \times FI \times CF \times EF \times EDa}{BWa \times ATa \times 365 \text{ days/yr}}$$

$$\text{INTAKE-CHILD} = \frac{CS \times IRc \times RAF \times FI \times CF \times EF \times EDc}{BWc \times ATc \times 365 \text{ days/yr}}$$

TABLE O-37, continued  
INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SETTLING POND 4  
RADGER ARMY AMMUNITION PLANT

SP-63308 23-Mar-93

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION KAF	INTAKE ADULT (mg/kg-day)	INTAKE CHILD (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK ADULT	CANCER RISK CHILD	TOTAL CANCER RISK
PB	300	1	1.4E-04	3.5E-04	ND			
SUMMARY CANCER RISK						0E+00	0E+00	0E+00

TABLE O-37, continued  
INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SETTLING POND 4  
BADGER ARMY AMMUNITION PLANT

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION FAF	INTAKE ADULT (mg/kg-day)	INTAKE CHILD (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT ADULT	HAZARD QUOTIENT CHILD	TOTAL HAZARD QUOTIENT
PB	300	1	4.1E-04	3.8E-03	ND	1.76E-04	1.64E-03	1.42E-03
SN	77	1	1.1E-04	9.8E-04	6.0E-01	1.37E-04	1.28E-03	1.42E-03
MTT	10	1	1.4E-05	1.3E-04	1.0E-01			
MB3	940	1	1.3E-03	1.3E-02	ND			
SO4	400	1	5.3E-04	3.1E-03	ND			
AL	60000	1	8.2E-02	7.7E-01	ND			
MC	1036	1	1.4E-03	1.3E-02	ND			
SUMMARY HAZARD INDEX						0.0003	0.0029	0.0032



TABLE O-38, continued

INCIDENTAL INGESTION AND INHALATION OF SOIL  
 GROUNDS MAINTENANCE WORKER  
 SETTLING POND 4  
 BADGER ARMY AMMUNITION PLANT

SPASSK1W 08 - Dec - 92

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RPF	INGESTION (mg/kg-day)	INTAKE INHALATION (mg/kg-day)	CANCER SLOPE FACTOR-INH. (mg/kg-day) <sup>-1</sup>	CANCER SLOPE FACTOR-ING. (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK INHALATION	TOTAL CANCER RISK
PB	300	1	1.0E-05	4.3E-10	ND				
SUMMARY CANCER RISK									0E+00

TABLE O-38, continued

INCIDENTAL INGESTION AND INHALATION OF SOIL  
 GROUNDS MAINTENANCE WORKER  
 SETTLING POND 4  
 BADGER ARMY AMMUNITION PLANT

SP4SSOW 08-Dec-92

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION BAF	INTAKE INGESTION (mg/kg-day)	AIR CONCENT. (mg/m <sup>3</sup> )	REFERENCE CONCENT. (mg/m <sup>3</sup> )	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT INHALATION	TOTAL HAZARD QUOTIENT
PB	300	1	2.8E-03	6.5E-08	ND	ND	ND		1.21E-05
SN	77	1	7.2E-06	1.7E-08	ND	6.0E-01	1.21E-05		9.39E-06
NT	10	1	9.4E-07	2.2E-09	ND	1.0E-01	9.39E-06		
NH3	960	1	9.0E-05	2.1E-07	ND	ND	ND		
SO4	400	1	3.8E-05	8.6E-08	ND	ND	ND		
AL	60000	1	5.6E-03	1.3E-05	ND	ND	ND		
NC	1038	1	9.8E-05	2.2E-07	ND	ND	ND		
SUMMARY HAZARD INDEX									0.00002
									0.00000
									0.00002

**Table O-39**  
**Compounds Detected**  
**Spoils Disposal Site 1**  
**Settling Ponds and Spoils Disposal Area Surface Soil (0-2')**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	<u>Concentration **</u>
AL	5 : 5	44258	12487	N	1	
FE	5 : 5	35401	4162	N	1, 3	
PB	5 : 5	349	42	Y		349
K	5 : 5	1660	55	N	1, 3	
NA	5 : 5	199	90	N	1, 3	
SN	5 : 5	3.68	2.54	Y		3.68
ZN	5 : 5	212	63	Y		212
BR	2 : 2	12		Y		12
CL	5 : 5	19	13	Y		19
NIT	5 : 5	16	8	Y		16
SO4	5 : 5	146	33	Y		146
CH2CL2	3 : 3	0.01	0.034	Y		0.01
24DNT	3 : 3	12	0.51	Y		12
26DNT	1 : 1	1		Y		1
B2EHP	1 : 1	0.35		Y		0.35
DNBP	5 : 5	51	0.82	Y		51
DNOP	1 : 1	8.6		Y		8.6
DPA	4 : 4	24	0.34	Y		24
NC	5 : 5	11000	6000	Y		11000
NG	1 : 1	19		Y		19

**Footnotes:**

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential for human nutrition.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum

**Note:** Assessment of surface soil contamination (0 to 2 feet) was performed using samples SD1-1 through SD1-5.

TABLE O-40

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SPOILS DISPOSAL SITE 1  
RADGER ARMY AMMUNITION PLANT

RD/ISSM01 75 - Mar - 93

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Medium	mg/kg	USEPA, 1991
INGESTION RATE - ADULT	IRa	100	mg/day	USEPA, 1991
INGESTION RATE - CHILD	IRc	200	mg/day	USEPA, 1991
FRACTION INGESTED	FI	100%		Assumption
CONVERSION FACTOR	CF	0.000001	kg/mg	USEPA, 1991
BODY WEIGHT - ADULT	BWa	70	kg	USEPA, 1991
BODY WEIGHT - CHILD	BWc	15	kg	USEPA, 1991
EXPOSURE FREQUENCY	EF	350	days/year	USEPA, 1991
EXPOSURE DURATION - ADULT	EDa	24	years	USEPA, 1991
EXPOSURE DURATION - CHILD	EDc	6	years	USEPA, 1991
AVERAGING TIME	AT	70	years	USEPA, 1989
CANCER	ATc	24	years	USEPA, 1991
ADULT - NONCANCER	ATc	6	years	USEPA, 1991
CHILD - NONCANCER	RAF	1	unitless	USEPA, 1989
RELATIVE ABSORPTION FACTOR				

USEPA, 1989, Risk Assessment Guidelines for Superfund  
USEPA, 1991, Standard Default Exposure Factors

Note:

For noncarcinogenic effects: AT = ED

CANCER RISK = INTAKE (mg/kg-day) x CANCER SLOPE FACTOR (mg/kg-day)<sup>-1</sup>

HAZARD QUOTIENT = INTAKE (mg/kg-day) / REFERENCE DOSE (mg/kg-day)

INTAKE-ADULT =

$$CS \times IRa \times FI \times CF \times EF \times EDa$$

$$BWa \times ATa \times 365 \text{ days/yr}$$

INTAKE-CHILD =

$$CS \times IRc \times FI \times CF \times EF \times EDc$$

$$BWc \times ATc \times 365 \text{ days/yr}$$



TABLE O-40, continued  
INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SOILS DISPOSAL SITE 1  
BADGER ARMY AMMUNITION PLANT

SDJSSME 25-Mar-93

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAV	INTAKE		INTAKE		CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK		CANCER RISK		TOTAL CANCER RISK
			ADULT (mg/kg-day)	CHILD (mg/kg-day)	ADULT (mg/kg-day)	CHILD (mg/kg-day)		ADULT	CHILD	ADULT	CHILD	
2,4-DNT	12	1	5.8E-06	1.3E-05	6.8E-01	6.8E-01	6.8E-01	3.8E-06	8.9E-06	3.8E-06	8.9E-06	1.5E-05
2,6-DNT	1	1	4.7E-07	1.1E-06	6.8E-01	6.8E-01	6.8E-01	3.2E-07	7.5E-07	3.2E-07	7.5E-07	1.1E-06
FB	349	1	1.6E-04	3.8E-04	ND	ND	ND					
BZEP	0.35	1	1.6E-07	3.8E-07	1.4E-02	1.4E-02	1.4E-02	2.5E-09	5.4E-09	2.5E-09	5.4E-09	7.7E-09
CBZOL	0.034	1	1.6E-08	3.7E-08	7.5E-03	7.5E-03	7.5E-03	1.2E-10	2.8E-10	1.2E-10	2.8E-10	4.0E-10
SUMMARY CANCER RISK												
								4E-06	1E-05	4E-06	1E-05	1E-05

TABLE O-40, continued

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SPOILS DISPOSAL SITE 1  
BADGER ARMY AMMUNITION PLANT

## NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION EAF	INTAKE		REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT		HAZARD QUOTIENT	
			ADULT (mg/kg-day)	CHILD (mg/kg-day)		ADULT	CHILD	ADULT	CHILD
Pb	349	1	4.8E-04	4.5E-03	ND	8.40E-06	7.84E-05	8.68E-05	
Sn	3.68	1	5.0E-06	4.7E-05	6.0E-01	1.45E-03	1.36E-02	1.50E-02	
Zn	212	1	2.9E-04	2.7E-03	2.0E-01	ND			
Br	12	1	1.6E-05	1.5E-04	ND				
Cl	19	1	2.6E-05	2.4E-04	ND				
Ni	16	1	2.2E-05	2.0E-04	1.0E-01	2.19E-04	2.05E-03	2.26E-03	
SO4	146	1	2.0E-04	1.9E-03	ND				
CH3CH2	0.034	1	4.7E-08	4.3E-07	6.0E-02	7.76E-07	7.23E-06	8.02E-06	
2MONT	12	1	1.6E-05	1.5E-04	2.0E-03	8.22E-03	7.67E-02	8.49E-02	
2MONT	1	1	1.4E-06	1.3E-05	ND				
B2ETP	0.35	1	4.8E-07	4.5E-06	2.0E-02	2.40E-03	2.24E-04	2.48E-04	
DMP	51	1	7.0E-05	6.5E-04	1.0E-01	6.99E-04	6.52E-03	7.22E-03	
DNOP	8.6	1	1.2E-05	1.1E-04	2.0E-02	5.89E-04	5.50E-03	6.09E-03	
DPA	24	1	3.3E-05	3.1E-04	2.0E-02	1.64E-03	1.53E-02	1.70E-02	
NC	11000	1	1.5E-02	1.4E-01	ND				
NO	19	1	2.6E-05	2.4E-04	ND				
SUMMARY HAZARD INDEX									
						0.013	0.120	0.133	0.133

**INCIDENTAL INGESTION AND INHALATION OF SOIL  
GROUNDS MAINTENANCE WORKER  
SPOILS DISPOSAL AREA 1  
BADGER ARMY AMMUNITION PLANT**

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Medium	mg/kg	USEPA, 1991a
INGESTION RATE	IR	100	mg/day	Assumption
FRACTION INGESTED	FI	100%		
CONVERSION FACTOR	CF	0.000001	kg/mg	
BODY WEIGHT	BW	70	kg	USEPA, 1991a
EXPOSURE FREQUENCY	EF	24	days/year	USEPA, 1991a
EXPOSURE DURATION	ED	25	years	USEPA, 1991a
CONCENTRATION AIR PARTICULATES	CAp	Calculated	mg/m <sup>3</sup>	Appendix M
CONCENTRATION AIR VOLATILES	CAv	Calculated	mg/m <sup>3</sup>	USEPA, 1991b
VOLATILIZATION FACTOR	VF	Calculated	m <sup>3</sup> /kg	USEPA, 1991a
PARTICULATE EMISSION FACTOR	PEF	4.63E+09	mg/kg	Assumption
INHAALATION RATE	IhR	2.5	m <sup>3</sup> /hour	
EXPOSURE TIME	ET	8	hours/day	
AVERAGING TIME				
RELATIVE ABSORPTION FACTOR				
CANCER	AT	70	years	USEPA, 1989
NONCANCER	AT	25	years	USEPA, 1991a
	RAF	1	unitless	USEPA, 1989

CANCER RISK = INTAKE (mg/kg-day) x CANCER SLOPE FACTOR (mg/kg-day)<sup>-1</sup>

HAZARD QUOTIENT<sub>ingestion</sub> = INTAKE (mg/kg-day) / REFERENCE DOSE (mg/kg-day)

HAZARD QUOTIENT<sub>inhalation</sub> =  $\frac{\text{AIR CONCENTRATION (mg/m}^3\text{)}}{\text{REFERENCE CONCENTRATION (mg/m}^3\text{)}}$

INTAKE - INGESTION =  $\frac{\text{CS} \times \text{IR} \times \text{RAF} \times \text{FI} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$

INTAKE - INHALATION =  $\frac{(\text{CAp} + \text{CAv}) \times \text{IhR} \times \text{ET} \times \text{VF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$

AIR CONCENTRATION (mg/m<sup>3</sup>) = CAp + CAv

CAp = CS x I/P EF

CAv = CS x I/VF

Note:

For noncarcinogenic effects: AT = ED

USEPA, 1989. Risk Assessment Guidelines for Superfund - Part A

USEPA, 1990. Exposure Factors Handbook

USEPA, 1991a. Standard Default Exposure Factors

USEPA, 1989. Risk Assessment Guidelines for Superfund - Part A

USEPA, 1991b. Risk Assessment Guidelines for Superfund - Part B

USEPA, 1991a. Standard Default Exposure Factors

INCIDENTAL INGESTION AND INHALATION OF SOIL  
 GROUNDS MAINTENANCE WORKER  
 SOILS DISPOSAL AREA 1  
 BADGER ARMY AMMUNITION PLANT

## CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION BAF	INTAKE INGESTION (mg/kg-day)	INTAKE INHALATION (mg/kg-day)	CANCER SLOPE FACTOR-ING. (mg/kg-day) <sup>-1</sup>	CANCER SLOPE FACTOR-INO. (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK INHALATION	TOTAL CANCER RISK
24DNT	12	1	4.0E-07	1.7E-11	ND	6.8E-01	2.7E-07		2.7E-07
24DNT	1	1	3.4E-08	1.4E-12	ND	6.8E-01	2.3E-08		2.3E-08
PB	349	1	1.2E-05	5.1E-10	ND	ND			
B2EIP	0.35	1	1.2E-08	5.1E-13	ND	1.4E-02	1.6E-10		1.6E-10
CH2CL2	0.034	1	1.1E-09	4.9E-14	ND	7.5E-03	8.6E-12		8.6E-12
SUMMARY CANCER RISK									
							3E-07	0E+00	3E-07

INCIDENTAL INGESTION AND INHALATION OF SOIL  
 GROUND MAINTENANCE WORKER  
 SPOILS DISPOSAL AREA 1  
 BADGER ARMY AMMUNITION PLANT

## NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION R/F	INTAKE (mg/kg-day)	AIR CONCENT. (mg/m <sup>3</sup> )	REFERENCE CONCENT. (mg/m <sup>3</sup> )	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT INHALATION	TOTAL HAZARD QUOTIENT
PB	349	1	3.3E-05	7.5E-08	ND	ND	5.76E-07		5.76E-07
SN	3.68	1	3.5E-07	7.9E-10	ND	6.0E-01	9.96E-05		9.96E-05
ZN	212	1	2.0E-05	4.6E-08	ND	2.0E-01			
BR	12	1	1.1E-06	2.6E-09	ND	ND			
CL	19	1	1.8E-06	4.1E-09	ND	ND			
NIT	16	1	1.5E-06	3.5E-09	ND	1.0E-01	1.50E-05		1.50E-05
SO4	146	1	1.4E-05	3.2E-08	ND	ND			
CH2CL2	0.034	1	3.2E-09	7.3E-12	ND	6.0E-02	5.32E-08		5.32E-08
24DNT	12	1	1.1E-06	2.6E-09	ND	2.0E-03	5.64E-04		5.64E-04
24DNT	1	1	9.4E-08	2.2E-10	ND	ND			
B2BIP	0.35	1	3.3E-08	7.6E-11	ND	2.0E-02	1.44E-06		1.44E-06
DNBP	51	1	4.8E-06	1.1E-08	ND	1.0E-01	4.79E-05		4.79E-05
DNOP	8.6	1	8.1E-07	1.9E-09	ND	2.0E-02	4.04E-05		4.04E-05
DPA	24	1	2.3E-06	5.2E-09	ND	2.0E-02	1.13E-04		1.13E-04
NC	11000	1	1.0E-03	2.4E-06	ND	ND			
NO	19	1	1.8E-06	4.1E-09	ND	ND			
SUMMARY HAZARD INDEX							0.0009	0.0000	0.0009

**Table O-42**  
**Compounds Detected**  
**Spoils Disposal Site 2**  
**Settling Ponds and Spoils Disposal Area Surface Soil (0-2')**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
AL	5: 5	49398	4547	N	1	
FE	5: 5	18674	15534	N	1, 3	
PB	5: 5	373	239	Y		373
K	5: 5	566	437	N	1, 3	
NA	5: 5	235	123	N	1, 3	
SN	5: 5	4.04	1.04	Y		4.04
ZN	5: 5	748	148	Y		748
BR	1: 1	4		Y		4
CL	5: 5	23	16	Y		23
NIT	5: 5	10	8	Y		10
SO4	5: 5	130	80	Y		130
CH2CL2	3: 3	0.012	0.024	Y		0.012
24DNT	4: 4	1.3	0.48	Y		1.3
DNBP	5: 5	5.8	0.98	Y		5.8
DPA	5: 5	3.2	0.24	Y		3.2
NC	5: 5	8000	5800	Y		8000

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant.  
                   \* 3 = essential for human nutrition.  
                   \* 4 = frequency of detection less than 5 %.  
                   \*\* 95th percentile or maximum

Note:           Assessment of surface soil contamination (0 to 2 feet) was performed using samples SD2-1 through SD2-5.

TABLE O-43

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SPOILS DISPOSAL SITE 2  
BADGER ARMY AMMUNITION PLANT

SD-253308

25-Mar-95

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Maximum	mg/kg	USEPA, 1991
INGESTION RATE - ADULT	IRa	100	mg/day	USEPA, 1991
INGESTION RATE - CHILD	IRc	200	mg/day	USEPA, 1991
FRACTION INGESTED	FI	100%		Assumption
CONVERSION FACTOR	CF	0.000001	kg/mg	USEPA, 1991
BODY WEIGHT - ADULT	BW <sub>a</sub>	70	kg	USEPA, 1991
BODY WEIGHT - CHILD	BW <sub>c</sub>	15	kg	USEPA, 1991
EXPOSURE FREQUENCY	EF	350	days/year	USEPA, 1991
EXPOSURE DURATION - ADULT	ED <sub>a</sub>	24	years	USEPA, 1991
EXPOSURE DURATION - CHILD	ED <sub>c</sub>	6	years	USEPA, 1991
AVERAGING TIME				
CANCER	AT	70	years	USEPA, 1989
ADULT - NONCANCER	AT <sub>a</sub>	24	years	USEPA, 1991
CHILD - NONCANCER	AT <sub>c</sub>	6	years	USEPA, 1991
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	USEPA, 1989

USEPA, 1989. Risk Assessment Guidance for Superfund  
USEPA, 1991. Standard Default Exposure Factors

Note:  
For noncarcinogenic effects: AT = ED

$$\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$$

$$\text{HAZARD QUOTIENT} = \text{INTAKE (mg/kg-day)} / \text{REFERENCE DOSE (mg/kg-day)}$$

$$\text{INTAKE-ADULT} = \frac{CS \times IRa \times RAF \times FI \times CF \times EF \times EDa}{BWa \times ATa \times 365 \text{ days/yr}}$$

$$\text{INTAKE-CHILD} = \frac{CS \times IRc \times RAF \times FI \times CF \times EF \times EDc}{BWc \times ATc \times 365 \text{ days/yr}}$$

INCIDENTAL INGESTION OF SURFACE SOIL,  
RESIDENTIAL - ADULT AND CHILD  
SPOILS DISPOSAL, SITE 2  
BADGER ARMY AMMUNITION PLANT

SD/SS/08

25-Mar-93

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAI	INTAKE ADULT (mg/kg-day)	INTAKE CHILD (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK ADULT	CANCER RISK CHILD	TOTAL CANCER RISK
ACONT	1.3	1	6.1E-07	1.4E-06	6.8E-01	4.2E-07	9.7E-07	1.4E-06
PB	373	1	1.4E-04	4.1E-04	ND			
CBDC12	0.024	1	1.1E-08	2.6E-08	7.5E-03	8.5E-11	2.0E-10	2.8E-10
SUMMARY CANCER RISK								
						4E-07	1E-06	1E-06



TABLE O-43, continued

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SOILS DISPOSAL SITE 2  
BADGER ARMY AMMUNITION PLANT

## NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION BAF	INTAKE		REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT		TOTAL HAZARD QUOTIENT
			ADULT (mg/kg-day)	CHILD (mg/kg-day)		ADULT	CHILD	
Pb	373	1	5.1E-04	4.8E-03	ND			
Sn	404	1	5.5E-06	5.2E-05	6.0E-01	9.22E-06	8.61E-05	9.53E-05
Zn	748	1	1.0E-03	9.6E-03	2.0E-01	5.12E-03	4.78E-02	5.29E-02
Br	4	1	5.5E-06	5.1E-05	ND			
Cl	23	1	3.2E-05	2.9E-04	ND			
Ni	10	1	1.4E-05	1.3E-04	1.0E-01	1.37E-04	1.28E-03	1.42E-03
SO4	130	1	1.8E-04	1.7E-03	ND			
CERCL3	0.024	1	3.3E-06	3.1E-07	6.0E-02	5.48E-07	5.11E-06	5.66E-06
240NT	1.3	1	1.8E-06	1.7E-05	2.0E-03	8.90E-04	8.31E-03	9.20E-03
DNEP	58	1	7.9E-06	7.4E-05	1.0E-01	7.95E-05	7.42E-04	8.21E-04
DPA	3.2	1	4.4E-06	4.1E-05	2.5E-02	1.75E-04	1.64E-03	1.81E-03
NC	8000	1	1.1E-02	1.0E-01	ND			
SUMMARY HAZARD INDEX						0.004	0.049	0.064

INCIDENTAL INGESTION AND INHALATION OF SOIL  
 GROUNDS MAINTENANCE WORKER  
 SPOOLS DISPOSAL AREA 2  
 BADGER ARMY AMMUNITION PLANT

SDZSSGJW 08 Dec-92

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Medium	mg/kg	USEPA, 1991a
INGESTION RATE	IR	100	mg/day	Assumption
FRACTION INGESTED	FI	100%		
CONVERSION FACTOR	CF	0.000001		
BODY WEIGHT	BW	70	kg	USEPA, 1991a
EXPOSURE FREQUENCY	EF	24	days/year	USEPA, 1991a
EXPOSURE DURATION	ED	25	years	USEPA, 1991a
CONCENTRATION AIR PARTICULATES	CAp	Calculated	mg/m <sup>3</sup>	Appendix M
CONCENTRATION AIR VOLATILES	CAv	Calculated	mg/m <sup>3</sup>	USEPA, 1991b
VOLATILIZATION FACTOR	VF	Calculated	m <sup>3</sup> /kg	USEPA, 1991a
PARTICULATE EMISSION FACTOR	PEF	4.63E+09	m <sup>3</sup> /hour	Assumption
INHALATION RATE	IR	2.5	m <sup>3</sup> /hour	
EXPOSURE TIME	ET	8	hours/day	
AVERAGING TIME	AT	70	years	USEPA, 1989
	AT	25	years	USEPA, 1991a
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	USEPA, 1989
CANCER NONCANCER RELATIVE ABSORPTION FACTOR				
USEPA, 1989. Risk Assessment Guidelines for Superfund - Part A USEPA, 1990. Exposure Factors Handbook USEPA, 1991a. Standard Default Exposure Factors				
USEPA, 1991b. Risk Assessment Guidelines for Superfund - Part B				

$$\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$$

$$\text{HAZARD QUOTIENT}_{\text{Ingestion}} = \text{INTAKE (mg/kg-day)} / \text{REFERENCE DOSE (mg/kg-day)}$$

$$\text{HAZARD QUOTIENT}_{\text{Inhalation}} = \frac{\text{AIR CONCENTRATION (mg/m}^3\text{)}}{\text{REFERENCE CONCENTRATION (mg/m}^3\text{)}}$$

$$\text{INTAKE-INGESTION} = \frac{\text{CS} \times \text{IR} \times \text{FI} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

$$\text{INTAKE-INHALATION} = \frac{(\text{CAp} + \text{CAv}) \times \text{IR} \times \text{VF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

$$\text{AIR CONCENTRATION (mg/m}^3\text{)} = \text{CAp} + \text{CAv}$$

$$\text{CAp} = \text{CS} \times \text{I/REF}$$

$$\text{CAv} = \text{CS} \times \text{V/VT}$$

Note:

For noncarcinogenic effects: AT = ED

TABLE O-44, continued

INCIDENTAL INGESTION AND INHALATION OF SOIL  
 GROUNDS MAINTENANCE WORKER  
 SPILLS DISPOSAL AREA 2  
 BADGER ARMY AMMUNITION PLANT

SDZSGJW 08-11-92

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION DAP	INTAKE INGESTION (mg/kg-dw)	INTAKE INHALATION (mg/kg-dw)	CANCER SLOPE FACTOR-DNE (mg/kg-dw) <sup>-1</sup>	CANCER SLOPE FACTOR-INO (mg/kg-dw) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK INHALATION	TOTAL CANCER RISK
24DNT	1.3	1	4.4E-08	1.9E-12	ND	6.8E-01	3.0E-08		3.0E-08
PB	373	1	1.3E-05	5.4E-10	ND	ND			
CH2CL2	0.024	1	8.1E-10	3.5E-14	ND	7.5E-03	6.0E-12		6.0E-12
SUMMARY CANCER RISK									
							3E-08	0E+00	3E-08

Table O-44, continued

INCIDENTAL INGESTION AND INHALATION OF SOIL  
GROUNDS MAINTENANCE WORKER  
SPOILS DISPOSAL AREA 2  
BADGER ARMY AMMUNITION PLANT

SD25SGW 08-10-92

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION LAP	INTAKE INGESTION (mg/kg-day)	AIR CONCENT. (mg/m <sup>3</sup> )	REFERENCE CONCENT. (mg/m <sup>3</sup> )	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT INHALATION	TOTAL HAZARD QUOTIENT
Pb	373	1	3.5E-05	8.1E-08	ND	ND	6.32E-07		6.32E-07
Sn	4.04	1	3.8E-07	8.7E-10	ND	6.0E-01	3.51E-04		3.51E-04
Zn	748	1	7.0E-05	1.6E-07	ND	2.0E-01			
Br	4	1	3.8E-07	8.6E-10	ND	ND			
Cl	23	1	2.2E-06	5.0E-09	ND	ND			
Ni	10	1	9.4E-07	2.2E-09	ND	1.0E-01	9.39E-06		9.39E-06
SO4	130	1	1.2E-05	2.8E-08	ND	ND			
CH2CL2	0.024	1	2.3E-09	5.2E-12	ND	6.0E-02	3.76E-08		3.76E-08
2,4-DNT	1.3	1	1.2E-07	2.8E-10	ND	2.0E-03	6.11E-05		6.11E-05
DMBP	5.8	1	5.4E-07	1.3E-09	ND	1.0E-01	5.45E-06		5.45E-06
DPA	3.2	1	3.0E-07	6.9E-10	ND	2.0E-02	1.50E-05		1.50E-05
NC	8000	1	7.5E-04	1.7E-06	ND	ND			
NO	19	1	1.8E-06	4.1E-09	ND	ND			
SUMMARY HAZARD INDEX							0.0004	0.0000	0.0004

**Table O-45**  
**Compounds Detected**  
**Spoils Disposal Site 3**  
**Settling Ponds and Spoils Disposal Area Surface Soil (0-2')**  
**Units ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
AL	10: 10	26530	7123	N	1	
FE	10: 10	15696	5224	N	1, 3	
PB	10: 10	67	24	Y		67
K	10: 10	1327	121	N	1, 3	
NA	10: 10	286	95	N	1, 3	
SN	10: 10	5.8	1.16	Y		5.8
ZN	10: 10	251	84	Y		251
CL	10: 10	17	10	Y		17
NIT	10: 10	22	9	Y		22
SO4	10: 10	75	29	Y		75
CH2CL2	1: 1	0.025		Y		0.025
24DNT	5: 5	1.1	0.24	Y		1.1
DNBP	9: 9	4	0.26	Y		4
DPA	5: 5	2.2	0.25	Y		2.2
NC	10: 10	3800	450	Y		3800

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant  
                   \* 3 = essential for human nutrition.  
                   \* 4 = frequency of detection less than 5 %.  
                   \*\* 95th percentile or maximum

Note:           Assessment of surface soil contamination (0 to 2 feet) was performed  
                   using samples from SD3-1 through SD3-10.

TABLE O-46

INCIDENTAL INGESTION OF SURFACE SOIL.  
RESIDENTIAL - ADULT AND CHILD  
SPOOLS DISPOSAL SITE 3  
BADGER ARMY AMMUNITION PLANT

SD 353506

23 - Mar - 93

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Median	mg/kg	USEPA, 1991
INGESTION RATE - ADULT	IRa	100	mg/day	USEPA, 1991
INGESTION RATE - CHILD	IRc	200	mg/day	USEPA, 1991
FACTION INGESTED	FI	100%		Assumption
CONVERSION FACTOR	CF	0.000001		
BODY WEIGHT - ADULT	BWa	70	kg	USEPA, 1991
BODY WEIGHT - CHILD	BWc	15	kg	USEPA, 1991
EXPOSURE FREQUENCY	EF	350	days/year	USEPA, 1991
EXPOSURE DURATION - ADULT	EDa	24	years	USEPA, 1991
EXPOSURE DURATION - CHILD	EDc	6	years	USEPA, 1991
AVERAGING TIME				
CANCER	AT	70	years	USEPA, 1989
ADULT - NONCANCER	ATa	24	years	USEPA, 1991
CHILD - NONCANCER	ATc	6	years	USEPA, 1991
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	USEPA, 1989

USEPA, 1989. Risk Assessment Guidelines for Superfund  
USEPA, 1991. Standard Default Exposure Factors

Note:  
For noncarcinogenic effects: AT = ED

$$\text{CANCER RISK} = \text{INTAKE} (\text{mg/kg-day}) \times \text{CANCER SLOPE FACTOR} (\text{mg/kg-day})^{-1}$$

$$\text{HAZARD QUOTIENT} = \text{INTAKE} (\text{mg/kg-day}) / \text{REFERENCE DOSE} (\text{mg/kg-day})$$

$$\text{INTAKE-ADULT} = \frac{CS \times IRa \times FI \times CF \times EF \times EDa}{BWa \times ATa \times 365 \text{ days/yr}}$$

$$\text{INTAKE-CHILD} = \frac{CS \times IRc \times FI \times CF \times EF \times EDc}{BWc \times ATc \times 365 \text{ days/yr}}$$

TABLE O-44, continued

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SPOOLS DISPOSAL SITE 3  
BADGER ARMY AMMUNITION PLANT

SDXSSM01 25-Mar-95

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAI	INTAKE		CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK		TOTAL CANCER RISK
			ADULT (mg/kg-day)	CHILD (mg/kg-day)		ADULT	CHILD	
ADONT	1.1	1	5.2E-07	1.2E-06	6.8E-01	3.5E-07	8.2E-07	1.2E-06
PO	67	1	3.1E-05	7.3E-05	ND			
CHROCL	0.025	1	1.2E-06	2.7E-06	7.5E-03	8.8E-11	2.1E-10	2.9E-10
SUMMARY CANCER RISK								
						4E-07	8E-07	1E-06

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SPOOLS DISPOSAL SITE 3  
BADGER ARMY AMMUNITION PLANT

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAI	INTAKE ADULT (mg/kg-day)	INTAKE CHILD (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT ADULT	HAZARD QUOTIENT CHILD	TOTAL HAZARD QUOTIENT
PB	67	1	9.2E-05	8.6E-04	ND	1.32E-03	1.24E-04	1.37E-04
SN	5.8	1	7.9E-06	7.4E-05	6.0E-01	1.72E-03	1.60E-02	1.70E-02
ZN	231	1	3.4E-04	3.2E-03	2.0E-01	ND	ND	ND
CL	17	1	2.3E-05	2.2E-04	1.0E-01	3.01E-04	2.81E-03	3.11E-03
NI	22	1	3.0E-05	2.8E-04	1.0E-01	ND	ND	ND
SO4	75	1	1.0E-04	9.6E-04	6.0E-02	5.71E-07	5.33E-06	5.90E-06
CH2CL2	0.025	1	3.4E-08	3.2E-07	2.0E-03	7.53E-04	7.03E-03	7.79E-03
ADMT	1.1	1	1.5E-06	1.4E-05	1.0E-01	5.48E-03	5.11E-04	5.66E-04
DIMP	4	1	5.3E-06	5.1E-05	2.5E-02	1.21E-04	1.13E-03	1.25E-03
DPA	2.2	1	3.0E-06	2.8E-05	ND	ND	ND	ND
MC	3000	1	5.2E-03	4.9E-02	ND	ND	ND	ND
SUMMARY HAZARD INDEX						0.003	0.028	0.031



TABLE O-47

INCIDENTAL INGESTION AND INHALATION OF SOIL.  
 GROUNDS MAINTENANCE WORKER  
 SPOILS DISPOSAL AREA 3  
 BADGER ARMY AMMUNITION PLANT

SIJ3SSOW

09 - Dec - 92

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Maximum	mg/kg	USEPA, 1991a
INGESTION RATE	IR	100	mg/day	Assumption
FRACTION INGESTED	FI	100%		
CONVERSION FACTOR	CF	0.000001	kg/mg	
BODY WEIGHT	BW	70	kg	USEPA, 1991a
EXPOSURE FREQUENCY	EF	24	days/year	USEPA, 1991a
EXPOSURE DURATION	ED	25	years	USEPA, 1991a
CONCENTRATION AIR PARTICULATES	CAp	Calculated	mg/m <sup>3</sup>	
CONCENTRATION AIR VOLATILES	CAV	Calculated	mg/m <sup>3</sup>	Appendix M
VOLATILIZATION FACTOR	VF	Calculated	m <sup>3</sup> /kg	USEPA, 1991b
PARTICULATE EMISSION FACTOR	PEF	4.63E+09	m <sup>3</sup> /hour	USEPA, 1991a
INHALATION RATE	IhR	2.5	m <sup>3</sup> /hour	Assumption
EXPOSURE TIME	ET	8	hours/day	
AVERAGING TIME	AT			
	AT	70	years	USEPA, 1989
	AT	25	years	USEPA, 1991a
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	USEPA, 1989

$$\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$$

$$\text{HAZARD QUOTIENT}_{\text{Ingestion}} = \text{INTAKE (mg/kg-day)} / \text{REFERENCE DOSE (mg/kg-day)}$$

$$\text{HAZARD QUOTIENT}_{\text{Inhalation}} = \frac{\text{AIR CONCENTRATION (mg/m}^3\text{)}}{\text{REFERENCE CONCENTRATION (mg/m}^3\text{)}}$$

$$\text{INTAKE-INGESTION} = \frac{\text{CS} \times \text{IR} \times \text{RAF} \times \text{FI} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

$$\text{INTAKE-INHALATION} = \frac{(\text{CAp} + \text{CAV}) \times \text{IhR} \times \text{ET} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

$$\text{AIR CONCENTRATION (mg/m}^3\text{)} = \text{CAp} + \text{CAV}$$

$$\text{CAp} = \text{CS} \times \text{VF} \times \text{PEF}$$

$$\text{CAV} = \text{CS} \times \text{IhR}$$

Note:

For noncarcinogenic effects: AT = ED

USEPA, 1989, Risk Assessment Guidelines for Superfund - Part A

USEPA, 1990, Exposure Factors Handbook

USEPA, 1991b, Risk Assessment Guidelines for Superfund - Part B

USEPA, 1991a, Standard Default Exposure Factors

INCIDENTAL INGESTION AND INHALATION OF SOIL  
 GROUNDS MAINTENANCE WORKER  
 SPOILS DISPOSAL AREA 3  
 BADGER ARMY AMMUNITION PLANT

SDSSSOW 09-100-92

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAI	INTAKE INGESTION (mg/kg-day)	INTAKE INHALATION (mg/kg-day)	CANCER SLOPE FACTOR-INH. (mg/kg-day) <sup>-1</sup>	CANCER SLOPE FACTOR-ING. (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK INHALATION	TOTAL CANCER RISK
24DNT PB	1.1	1	3.7E-08	1.4E-12	ND	6.8E-01	2.5E-08		2.5E-08
	67	1	2.2E-06	9.7E-11	ND	ND			
CIDCL2	0.025	1	8.4E-10	3.6E-14	ND	7.5E-03	6.3E-12		6.3E-12
SUMMARY CANCER RISK									3E-08
									0E+00

TABLE O-47, continued

SD35SGW 09-Dec-92

INCIDENTAL INGESTION AND INHALATION OF SOIL  
GROUNDS MAINTENANCE WORKER  
SPOILS DISPOSAL AREA 3  
BADGER ARMY AMMUNITION PLANT

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAF	INTAKE INGESTION (mg/kg-day)	AIR CONCENT. (mg/m <sup>3</sup> )	REFERENCE CONCENT. (mg/m <sup>3</sup> )	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT INHALATION	TOTAL HAZARD QUOTIENT
PB	67	1	6.3E-06	1.4E-08	ND	ND	9.08E-07		9.08E-07
SN	5.8	1	5.4E-07	1.3E-09	ND	6.0E-01	1.18E-04		1.18E-04
ZN	251	1	2.4E-05	5.4E-08	ND	2.0E-01			
CL	17	1	1.6E-06	3.7E-09	ND	ND			
MIT	22	1	2.1E-06	4.8E-09	ND	1.0E-01	2.07E-05		2.07E-05
SO4	75	1	7.0E-06	1.6E-08	ND	ND			
CH2CL2	0.025	1	2.3E-09	5.4E-12	ND	6.0E-02	3.91E-08		3.91E-08
24DNT	1.1	1	1.0E-07	2.4E-10	ND	2.0E-03	5.17E-05		5.17E-05
DNRIP	4	1	3.8E-07	8.6E-10	ND	1.0E-01	3.76E-06		3.76E-06
DPA	2.2	1	2.1E-07	4.8E-10	ND	2.0E-02	1.03E-05		1.03E-05
NC	3800	1	3.6E-04	8.2E-07	ND	ND			
SUMMARY HAZARD INDEX							0.0002	0.0000	0.0002

**Table O-48**  
**Compounds Detected**  
**Spoils Disposal Site 4**  
**Settling Ponds and Spoils Disposal Area Surface Soil (0-2')**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	<u>Concentration **</u>
AL	10 : 10	20865	11511	N	1	
FE	10 : 10	19894	13512	N	1, 3	
PB	10 : 10	120	22	Y		120
K	10 : 10	1819	415	N	1, 3	
NA	10 : 10	255	98	N	1, 3	
SN	10 : 10	1.64	0.63	Y		1.64
ZN	10 : 10	204	89	Y		204
CL	9 : 9	13	10	Y		13
NIT	10 : 10	12	4	Y		12
SO4	10 : 10	139	22	Y		139
CH2CL2	4 : 4	0.01	0.038	Y		.01
24DNT	1 : 1	0.7		Y		.7
B2EHP	1 : 1	0.32		Y		.32
DNBP	4 : 4	4.4	0.32	Y		4.4
DNOP	3 : 3	0.63	0.22	Y		0.63
DPA	1 : 1	1.1		Y		1.1
NC	9 : 9	3000	33	Y		3000

Footnotes:

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential for human nutrition.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum

Note: Assessment of surface soil contamination (0 to 2 feet) was performed using samples from SD4-1 through SD4-10.

TABLE O-49

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SPOILS DISPOSAL SITE 4  
RADGER ARMY AMMUNITION PLANT

SD-K3586 25-Mar-91

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Median	mg/kg	USEPA 1991
INGESTION RATE - ADULT	IRa	100	mg/day	USEPA 1991
INGESTION RATE - CHILD	IRc	200	mg/day	USEPA 1991
FRACTION INGESTED	FI	100%		Assumption
CONVERSION FACTOR	CF	0.000001	kg/mg	USEPA 1991
BODY WEIGHT - ADULT	BW <sub>a</sub>	70	kg	USEPA 1991
BODY WEIGHT - CHILD	BW <sub>c</sub>	15	kg	USEPA 1991
EXPOSURE FREQUENCY	EF	350	days/year	USEPA 1991
EXPOSURE DURATION - ADULT	ED <sub>a</sub>	24	years	USEPA 1991
EXPOSURE DURATION - CHILD	ED <sub>c</sub>	6	years	USEPA 1991
AVERAGING TIME	AT	70	years	USEPA 1989
ADULT - NONCANCER	AT <sub>a</sub>	24	years	USEPA 1991
CHILD - NONCANCER	AT <sub>c</sub>	6	years	USEPA 1991
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	USEPA 1989

<p>CANCER RISK = INTAKE (mg/kg-day) × CANCER SLOPE FACTOR (mg/kg-day)<sup>-1</sup> - 1</p> <p>HAZARD QUOTIENT = INTAKE (mg/kg-day) / REFERENCE DOSE (mg/kg-day)</p> <p>INTAKE-ADULT = <math>\frac{CS \times IR_a \times FI \times CF \times EF \times ED_a}{BW_a \times AT_a \times 365 \text{ days/yr}}</math></p> <p>INTAKE-CHILD = <math>\frac{CS \times IR_c \times FI \times CF \times EF \times ED_c}{BW_c \times AT_c \times 365 \text{ days/yr}}</math></p>	<p>Note:</p> <p>For noncarcinogenic effects: AT = ED</p>
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USEPA 1989, Risk Assessment Guidelines for Superfund  
USEPA 1991, Standard Default Exposure Factors

TABLE O-49, continued  
INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SPORTS DISPOSAL SITE 4  
BADGER ARMY AMMUNITION PLANT

## CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RPF	INTAKE (mg/kg-day)		CANCER FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK		TOTAL CANCER RISK
			ADULT	CHILD		ADULT	CHILD	
ADMT	0.7	1	3.3E-07	7.7E-07	6.8E-01	2.2E-07	5.2E-07	7.5E-07
FB	120	1	5.6E-03	1.3E-04	ND			
BZBP	0.32	1	1.5E-07	3.5E-07	1.4E-02	2.1E-09	4.9E-09	7.0E-09
CBZCLJ	0.038	1	1.8E-08	4.2E-08	7.5E-03	1.3E-10	3.1E-10	4.5E-10
SUMMARY CANCER RISK						2E-07	5E-07	8E-07

TABLE O-49, continued  
INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SPOOLS DISPOSAL SITE 4  
BADGER ARMY AMMUNITION PLANT

## NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION EAF	INTAKE ADULT (mg/kg-day)	INTAKE CHILD (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT ADULT	HAZARD QUOTIENT CHILD	TOTAL HAZARD QUOTIENT
PB	120	1	1.0E-04	1.3E-03	ND	3.74E-06	3.49E-05	3.87E-05
SN	1.64	1	2.2E-06	2.1E-03	6.0E-01	1.40E-03	1.30E-02	1.44E-02
ZN	204	1	2.8E-04	2.6E-03	2.0E-01	1.40E-03	1.30E-02	1.44E-02
CL	13	1	1.8E-03	1.7E-04	ND	1.64E-04	1.53E-03	1.70E-03
MT	12	1	1.8E-03	1.3E-04	1.0E-01	1.64E-04	1.53E-03	1.70E-03
SO4	139	1	1.9E-04	1.8E-03	ND	8.68E-07	8.10E-06	8.96E-06
CECCL3	0.038	1	3.2E-06	4.9E-07	6.0E-02	4.79E-04	4.47E-03	4.93E-03
ADMT	0.7	1	9.6E-07	8.9E-06	2.0E-03	2.19E-03	2.05E-04	2.38E-04
BZEP	0.32	1	4.4E-07	4.1E-06	2.0E-02	6.03E-05	5.63E-04	6.23E-04
DHEP	4.4	1	6.0E-06	3.6E-03	1.0E-01	4.32E-05	4.03E-04	4.46E-04
DNOP	0.43	1	8.6E-07	8.1E-06	2.0E-02	6.03E-05	5.63E-04	6.23E-04
DPA	1.1	1	1.3E-06	1.4E-05	2.5E-02	6.03E-05	5.63E-04	6.23E-04
HC	3000	1	4.1E-03	3.8E-02	ND			
SUMMARY HAZARD INDEX						0.8022	0.0206	0.8231





TABLE O - 50, continued

INCIDENTAL INGESTION AND INHALATION OF SOIL  
 GROUNDS MAINTENANCE WORKER  
 SOILS DISPOSAL AREA 4  
 BADGER ARMY AMMUNITION PLANT

SD4SSGW 09 Dec-92

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION BAF	INTAKE INGESTION (mg/kg-day)	INTAKE INHALATION (mg/kg-day)	CANCER SLOPE FACTOR-DNE (mg/kg-day) <sup>-1</sup>	CANCER SLOPE FACTOR-ING. (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK INHALATION	TOTAL CANCER RISK
24DNT	0.7	1	2.3E-08	1.0E-12	ND	6.8E-01	1.6E-08		1.6E-08
Pb	120	1	4.0E-06	1.7E-10	ND	ND			1.5E-10
B2EHP	0.32	1	1.1E-08	4.6E-13	ND	1.4E-02	1.5E-10		1.5E-10
CH2CL2	0.038	1	1.3E-09	5.5E-14	ND	7.5E-03	9.6E-12		9.6E-12
SUMMARY CANCER RISK									
							2E-08	0E+00	2E-08

INCIDENTAL INGESTION AND INHALATION OF SOIL.  
 GROUNDS MAINTENANCE WORKER  
 SOILS DISPOSAL AREA 4  
 BADGER ARMY AMMUNITION PLANT

## NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION EAF	INTAKE INGESTION (mg/kg-day)	AIR CONCENT. (mg/m <sup>3</sup> )	REFERENCE CONCENT. (mg/m <sup>3</sup> )	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT INHALATION	TOTAL HAZARD QUOTIENT
PB	120	1	1.1E-03	2.8E-08	ND	ND	2.57E-07		2.57E-07
SN	1.64	1	1.5E-07	3.5E-10	ND	6.0E-01	9.58E-05		9.58E-05
ZN	204	1	1.9E-05	4.4E-08	ND	2.0E-01	ND		ND
CL	13	1	1.2E-06	2.8E-09	ND	1.0E-01	1.13E-05		1.13E-05
MT	12	1	1.1E-06	2.8E-09	ND	ND	5.95E-08		5.95E-08
SO4	139	1	1.3E-05	3.0E-08	ND	6.0E-02	3.29E-05		3.29E-05
CH3CL2	0.038	1	3.6E-09	8.2E-12	ND	2.0E-03	1.50E-06		1.50E-06
24DNT	0.7	1	6.6E-08	1.5E-10	ND	1.0E-01	4.13E-06		4.13E-06
B2EIP	0.32	1	3.0E-08	6.9E-11	ND	2.0E-02	2.96E-06		2.96E-06
DNBP	4.4	1	4.1E-07	9.5E-10	ND	2.0E-02	5.17E-06		5.17E-06
DNOP	0.63	1	5.9E-08	1.4E-10	ND	ND			
DPA	1.1	1	1.0E-07	2.4E-10	ND	ND			
NC	3000	1	2.8E-04	6.5E-07	ND	ND			
SUMMARY HAZARD INDEX							0.0002	0.0000	0.0002

**Table O-51**  
**Compounds Detected**  
**Spoils Disposal Site 5**  
**Settling Ponds and Spoils Disposal Area Surface Soil (0-2')**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason</u>	
AL	9 : 9	19436	3684	N	1	
FE	9 : 9	18922	10591	N	1, 3	
PB	8 : 8	102	23	Y		102
K	9 : 9	1336	111	N	1, 3	
NA	10 : 10	216	64	N	1, 3	
SN	10 : 10	1.94	0.63	Y		1.94
ZN	9 : 9	306	101	Y		306
BR	1 : 1	16		Y		16
CL	9 : 9	18	10	Y		18
NIT	10 : 10	18	7	Y		18
SO4	10 : 10	38	23	Y		38
CH2CL2	3 : 3	0.01	0.026	Y		.01
DNBP	7 : 7	6.5	0.33	Y		6.5
DNOP	1 : 1	0.2		Y		.2
DPA	3 : 3	2.4	0.22	Y		2.4
NC	8 : 8	11000	250	Y		11000

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant.  
                   \* 3 = essential for human nutrition.  
                   \* 4 = frequency of detection less than 5 %.  
                   \*\* 95th percentile or maximum

Note:           Assessment of surface soil contamination (0 to 2 feet) was performed using samples from SD5-1 through SD5-10.

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SPOILS DISPOSAL SITE 5  
BADGER ARMY AMMUNITION PLANT

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Medium	mg/kg	USEPA 1991
INGESTION RATE - ADULT	IRa	100	mg/day	USEPA 1991
INGESTION RATE - CHILD	IRc	200	mg/day	USEPA 1991
FRACTION INGESTED	FI	100%		Assumption
CONVERSION FACTOR	CF	0.000001	kg/mg	USEPA 1991
BODY WEIGHT - ADULT	BWa	70	kg	USEPA 1991
BODY WEIGHT - CHILD	BWc	15	kg	USEPA 1991
EXPOSURE FREQUENCY	EF	350	days/year	USEPA 1991
EXPOSURE DURATION - ADULT	EDa	24	years	USEPA 1991
EXPOSURE DURATION - CHILD	EDc	6	years	USEPA 1991
AVERAGING TIME				
CANCER	AT	70	years	USEPA 1989
ADULT - NONCANCER	ATa	24	years	USEPA 1991
CHILD - NONCANCER	ATc	6	years	USEPA 1991
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	USEPA 1989

USEPA 1989. Risk Assessment Guidelines for Superfund  
USEPA 1991. Standard Default Exposure Factors

Note:  
For noncarcinogenic effects: AT = ED

$$\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$$

$$\text{HAZARD QUOTIENT} = \text{INTAKE (mg/kg-day)} / \text{REFERENCE DOSE (mg/kg-day)}$$

$$\text{INTAKE-ADULT} = \frac{CS \times IRa \times FI \times CF \times EF \times EDa}{BWa \times ATa \times 365 \text{ days/yr}}$$

$$\text{INTAKE-CHILD} = \frac{CS \times IRc \times FI \times CF \times EF \times EDc}{BWc \times ATc \times 365 \text{ days/yr}}$$

TABLE O-52, continued  
INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SPOB'S DISPOSAL SITE 5  
BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RPF	INTAKE ADULT (mg/kg-day)	INTAKE CHILD (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK ADULT	CANCER RISK CHILD	TOTAL CANCER RISK
PB	102	1	4.8E-05	1.1E-04	ND			
CHROCL	0.026	1	1.2E-06	2.8E-06	7.5E-03	9.2E-11	2.1E-10	3.1E-10
SUMMARY CANCER RISK						9E-11	2E-10	3E-10

TABLE O-52, continued

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
SPOILS DISPOSAL SITE 5  
BADGER ARMY AMMUNITION PLANT

## NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAV	INTAKE		REFERENCE DOSE (mg/kg-dm)	HAZARD QUOTIENT		TOTAL HAZARD QUOTIENT
			ADULT (mg/kg-dm)	CHILD (mg/kg-dm)		ADULT	CHILD	
Pb	102	1	1.4E-04	1.3E-03	ND			
Sn	1.94	1	2.7E-06	2.5E-03	6.0E-01	4.43E-06	4.13E-03	4.58E-03
Zn	306	1	4.2E-04	3.9E-03	2.0E-01	2.10E-03	1.96E-02	2.17E-02
Ba	16	1	2.2E-03	2.0E-04	ND			
Cl	18	1	2.5E-03	2.3E-04	ND			
Ni	18	1	2.5E-03	2.3E-04	1.0E-01	2.47E-04	2.30E-03	2.55E-03
So	36	1	5.2E-03	4.9E-04	ND			
CEXCL2	0.036	1	3.6E-08	3.3E-07	6.0E-02	5.94E-07	5.54E-06	6.13E-06
DHEP	6.3	1	8.9E-06	8.3E-03	1.0E-01	8.90E-03	8.31E-04	9.20E-04
DHOP	0.2	1	2.7E-07	2.6E-06	2.0E-02	1.37E-03	1.28E-04	1.42E-04
DFA	2.4	1	3.3E-06	3.1E-03	2.5E-02	1.32E-04	1.23E-03	1.36E-03
NC	1100	1	1.5E-02	1.4E-01	ND			
SUMMARY HAZARD INDEX						0.0026	0.0041	0.0267



TABLE O-53, continued

INCIDENTAL INGESTION AND INHALATION OF SOIL  
 GROUNDS MAINTENANCE WORKER  
 SPOILS DISPOSAL AREA 5  
 BADGER ARMY AMMUNITION PLANT

SDSSS/JW

09-188-52

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION BAF	INTAKE INGESTION (mg/kg-day)	INTAKE INHALATION (mg/kg-day)	CANCER SLOPE FACTOR-INEL (mg/kg-day) <sup>-1</sup>	CANCER SLOPE FACTOR-ING. (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK INHALATION	TOTAL CANCER RISK
PB CH2CL2	102 0.026	1 1	3.4E-06 8.7E-10	1.5E-10 3.8E-14	ND ND	7.5E-03 ND	6.5E-12		6.5E-12
SUMMARY CANCER RISK							7E-12	0E+00	7E-12

ABB Environmental Services, Inc.

Rev. R92



INCIDENTAL INGESTION AND INHALATION OF SOIL  
 GROUNDS MAINTENANCE WORKER  
 SPOILS DISPOSAL AREA 5  
 BADGER ARMY AMMUNITION PLANT

SDSSGOW

09 - Dec - 92

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION BAF	INTAKE INGESTION (mg/kg-day)	AIR CONCENT. (mg/m <sup>3</sup> )	REFERENCE CONCENT. (mg/m <sup>3</sup> )	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT INHALATION	TOTAL HAZARD QUOTIENT
Pb	102	1	9.6E-06	2.2E-08	ND	ND	3.04E-07		3.04E-07
Sn	1.94	1	1.8E-07	4.2E-10	ND	6.0E-01	1.44E-04		1.44E-04
Zn	306	1	2.9E-05	6.6E-08	ND	2.0E-01			
Hr	16	1	1.5E-06	3.5E-09	ND	ND			
Cl	18	1	1.7E-06	3.9E-09	ND	ND			
NTT	18	1	1.7E-06	3.9E-09	ND	1.0E-01	1.69E-05		1.69E-05
SO4	38	1	3.6E-06	8.2E-09	ND	ND			
CH2Cl2	0.026	1	2.4E-09	5.6E-12	ND	6.0E-02	4.07E-08		4.07E-08
DNBP	6.5	1	6.1E-07	1.4E-09	ND	1.0E-01	6.11E-06		6.11E-06
DNOP	0.2	1	1.9E-08	4.3E-11	ND	2.0E-02	9.39E-07		9.39E-07
DPA	2.4	1	2.3E-07	5.2E-10	ND	2.0E-02	1.13E-05		1.13E-05
NC	11000	1	1.0E-03	2.4E-06	ND	ND			
SUMMARY HAZARD INDEX							0.0002	0.0000	0.0002

Table O-54  
Compounds Detected  
Deterrent Burning Ground Subsurface Soil (2'-12')  
Units: ug/g

Remedial Investigation  
Badger Army Ammunition Plant

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	Retained for Risk Assessment		Exposure Point
				<u>(Y/N)?</u>	<u>Reason *</u>	<u>Concentration **</u>
24DNT	9 : 12	37000	16.8	Y		37000
26DNT	9 : 12	1400	2.11	Y		1400
3NT	4 : 12	6.9	1.52	Y		6.9
AS	4 : 12	7.88	3.01	Y		7.88
B2EHP	5 : 12	4.35	1.16	Y		4.35
C6H6	10 : 12	5.25	0.001	Y		5.25
CH2CL2	1 : 12	0.002	-	N	2	
CR	12 : 12	13.2	1.91	Y		13.2
CU	11 : 12	23.1	7.94	N	1	
DNBP	9 : 12	62	2.99	Y		62
FANT	1 : 12	0.139	-	Y		0.139
MEC6H5	1 : 12	0.138	-	Y		0.138
NI	11 : 12	10.2	3.34	Y		10.2
NIT	12 : 12	18.7	1.6	Y		18.7
NNDPA	12 : 12	2200	0.193	Y		2200
PB	15 : 15	20.2	2.61	N	1	
PHANTR	1 : 12	0.183	-	Y		0.183
PYR	1 : 12	0.144	-	Y		0.144
SO4	1 : 12	5.19	-	Y		5.19
TXYLEN	1 : 3	0.001	-	Y		0.001
ZN	12 : 12	26.7	6.35	Y		26.7

Footnotes:

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential for human nutrition.
- \* 4 = frequency of detection less than 5 %
- \*\* 95th percentile or maximum

Note: Assessment of subsurface soil contamination (2 to 12 feet) was performed using samples from borings DBB-91-01 through DBB-91-03.

TABLE O-55

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (2 - 12 feet)  
CONSTRUCTION WORKER  
DETERRENT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

DROSB-CW

08 - Dec - 92

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Maximum	mg/kg	USEPA, 1991
INGESTION RATE	IR	480	mg/day	Assumption
FRACTION INGESTED	FI	100%		USEPA, 1992
SOIL ADHERENCE FACTOR	SAF	1	mg/cm <sup>2</sup>	USEPA, 1990
SURFACE AREA EXPOSED	SA	2,100	cm <sup>2</sup> /day	USEPA, 1990
CONVERSION FACTOR	CF	0.000001	kg/mg	USEPA, 1991
BODY WEIGHT	BW	70	kg	Assumption
EXPOSURE FREQUENCY	EF	20	days/year	Assumption
EXPOSURE DURATION	ED	1	years	Assumption
AVERAGING TIME	AT	70	years	USEPA, 1989
CANCER	AT	0.0547943203	years	Assumption
NONCANCER	AT			
RELATIVE ABSORPTION FACTOR	RAF			USEPA, 1989
INGESTION				
DERMAL		1	unitless	USEPA, 1989

USEPA, 1989. Risk Assessment Guidelines for Superfund

USEPA, 1990. Exposure Factors Handbook

USEPA, 1991. Standard Default Exposure Factors

USEPA, 1992. Dermal Absorption Guidelines

$$\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$$

$$\text{HAZARD QUOTIENT} = \text{INTAKE (mg/kg-day)} / \text{REFERENCE DOSE (mg/kg-day)}$$

$$\text{INTAKE} = (\text{INTAKE-INGESTION}) + (\text{INTAKE-DERMAL})$$

$$\text{INTAKE-INGESTION} = \frac{CS \times IR \times FI \times SAF \times CF \times EF \times ED}{BW \times AT \times 365 \text{ days/yr}}$$

$$\text{INTAKE-DERMAL} = \frac{CS \times SA \times SAF \times RAF \times CF \times EF \times ED}{BW \times AT \times 365 \text{ days/yr}}$$

Note:

For noncarcinogenic effects: AT =

EF

365 days

## DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (2 - 12 feet)

## CONSTRUCTION WORKER

## DETERRENT BURNING GROUND

## BADGER ARMY AMMUNITION PLANT

## CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION BAF	INTAKE INGESTION (mg/kg-day)	DERMAL BAF	INTAKE DERMAL (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK DERMAL	TOTAL CANCER RISK
340MT	37000	1	2.0E-04	No values available	6.8E-01	1.4E-04	1.4E-04		1.4E-04
340MT	1400	1	7.5E-06		6.8E-01	5.1E-06	5.1E-06		5.1E-06
AS	7.88	1	4.2E-08	for	1.8E+00	7.6E-08	7.6E-08		7.6E-08
BZEP	4.35	1	2.3E-08	Quantitative	1.4E-02	3.3E-10	3.3E-10		3.3E-10
CB6	5.25	1	2.8E-08	Analysis	2.9E-02	8.2E-10	8.2E-10		8.2E-10
CR	13.2	1	7.1E-08		ND				
INDMBA	0.018	1	9.7E-11		5.1E+01	4.9E-09	4.9E-09		4.9E-09
INDPA	2200	1	1.2E-05		4.9E-03	5.8E-08	5.8E-08		5.8E-08
SUMMARY CANCER RISK							1E-04	8E+00	1E-04

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (2 - 12 feet)

CONSTRUCTION WORKER  
DETERGENT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

DIKISR-CW

08 Dec-92

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAP	INGESTION (mg/kg-day)	DERMAL RAP	DERMAL (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT DERMAL	TOTAL HAZARD QUOTIENT
13DNB	0.29	1	2.0E-06	No values available for Quantitative Analysis		4.0E+00	4.97E-07		4.97E-07
24DNB	3700	1	2.5E-01			ND			
26DNB	1400	1	9.6E-03			ND			
26DNAP	0.09	1	6.2E-07			4.0E-02	1.54E-05		1.54E-05
3NT	6.9	1	4.7E-05			1.0E-01	4.73E-04		4.73E-04
AS	7.88	1	5.4E-05			3.0E-04	1.80E-01		1.80E-01
BZDEP	4.35	1	3.0E-05			2.0E-02	1.49E-03		1.49E-03
CB66	5.25	1	3.6E-05			ND			
CR	13.2	1	9.1E-05			2.0E-02	4.53E-03		4.53E-03
DIP	25.9	1	1.8E-04			8.0E+00	2.22E-05		2.22E-05
DMAP	62	1	4.3E-04			1.0E+00	4.25E-04		4.25E-04
PAANT	0.139	1	9.5E-07			4.0E-01	2.38E-06		2.38E-06
MIBC65	0.138	1	9.5E-07			2.0E+00	4.73E-07		4.73E-07
ME	10.2	1	7.0E-05			2.0E-02	3.50E-03		3.50E-03
NET	18.7	1	1.3E-04			1.0E-01	1.28E-03		1.28E-03
NDME6A	0.018	1	1.2E-07			ND			
NDPA	2200	1	1.5E-02			ND			
PRANTR	0.183	1	1.3E-06			4.0E-02	3.14E-05		3.14E-05
PVR	0.144	1	9.9E-07			3.0E-01	3.29E-06		3.29E-06
SO4	5.19	1	3.6E-05			ND			
TXLYEN	0.002	1	1.4E-06			4.0E+00	3.43E-09		3.43E-09
ZN	26.7	1	1.8E-04			2.0E-01	9.15E-04		9.15E-04
SUMMARY HAZARD INDEX							0.1928	0.0000	0.1928

TABLE O-56

INITIATION EXPOSURE TO AMBIENT AIR  
CONSTRUCTION WORKER  
DETERRENT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

DSGJAW 09-Dec-92

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Maximum	mg/kg	@ zero - 12 feet
CONCENTRATION AIR PARTICULATES	CAp	Calculated	mg/m <sup>3</sup>	see below
CONCENTRATION AIR VOLATILES	CAv	Calculated	mg/m <sup>3</sup>	see below
VOLATILIZATION FACTOR	VF	Calculated	m <sup>3</sup> /kg	Appendix M
24 HOUR AVERAGE PM10 STANDARD	PM10	150	ug/m <sup>3</sup>	USEPA, 1991b
CONVERSION FACTOR	CF	1E-09	kg/hr	USEPA, 1991a
INHALATION RATE	IR	2.5	m <sup>3</sup> /hour	USEPA, 1991a
BODY WEIGHT	BW	70	kg	USEPA, 1989
EXPOSURE TIME	ET	8	hour/day	Assumption
EXPOSURE FREQUENCY	EF	20	day/year	Assumption
EXPOSURE DURATION	ED	1	years	Assumption
AVERAGING TIME	AT	70	years	USEPA, 1991a
CANCER	AT	0.0547945203	years	USEPA, 1991a
NON-CANCER	AT			
USEPA, 1989, Risk Assessment Guidance for Superfund, Part A				
USEPA, 1991a, Standard Default Exposure Factors				
USEPA, 1991b, CFB50495-097				

$$\text{CANCER RISK} = \text{INTAKE} (\text{mg/kg-day}) \times \text{CANCER SLOPE FACTOR} (\text{mg/kg-day})^{-1} - 1$$

$$\text{HAZARD QUOTIENT} = \text{AIR CONCENTRATION} (\text{mg/m}^3) / \text{REFERENCE CONCENTRATION} (\text{mg/m}^3)$$

$$\text{INTAKE} = \frac{(\text{CAp} + \text{CAv}) \times \text{IR} \times \text{ET} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

$$\text{AIR CONCENTRATION} (\text{mg/m}^3) = \text{CAp} + \text{CAv}$$

$$\text{CAp} = \text{CS} \times \text{PM10} \times \text{CF}$$

$$\text{CAv} = \text{CS} \times \text{V/F}$$

Note:

For noncarcinogenic effects: AT = 365 days

EF =

365 days

TABLE O-56, continued  
 INHALATION EXPOSURE TO AMBIENT AIR  
 CONSTRUCTION WORKER  
 DETERGENT BURNING GROUND  
 BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VP (mmHg)	AIR CONCENTRATION VOLATILES (mg/m <sup>3</sup> )	AIR CONCENTRATION PARTICULATES (mg/m <sup>3</sup> )	INTAKE (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK
ACENT	3700			0.0033	1.2E-06	ND	
ACENT	140			0.0021	4.7E-06	ND	
AS	7.88			0.00001182	2.6E-10	5.0E+01	1.3E-06
BZEMP	4.35			0.000000625	1.5E-10	ND	
CB64	5.25	4220	0.0012440738	0.000007875	2.8E-07	2.9E-02	8.1E-09
CR	13.2			0.00000198	4.4E-10	4.1E+01	1.8E-06
MSDPA	2200			0.0003	7.4E-06	ND	
SUMMARY CANCER RISK							4E-06

TABLE O-54, continued  
INITIALATION EXPOSURE TO AMBIENT AIR  
CONSTRUCTION WORKER  
DETERGENT BURNING GROUND  
BADGER ARMY AMMUNITION PLANT

DEQARW

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VF (m <sup>3</sup> /kg)	AIR CONCENTRATION VOLATILES (mg/m <sup>3</sup> )	AIR CONCENTRATION PARTICULATES (mg/m <sup>3</sup> )	REFERENCE CONCENTRATION (mg/m <sup>3</sup> )	HAZARD QUOTIENT VOLATILES	HAZARD QUOTIENT PARTICULATES	HAZARD QUOTIENT TOTAL
MERCURY	0.138	8010	0.000017285	0.000000007	2.0E+00	8.6E-06	1.0E-06	8.6E-06
No other CVDs have RfCs								
SUMMARY HAZARD INDEX						0.0000041	0.0000001	0.0000042



Table O-57  
Compounds Detected  
Rocket Paste Area Surface Soil (0-2')  
Units: ug/g

Remedial Investigation  
Badger Army Ammunition Plant

Compound	Frequency	Maximum	Minimum	Retained for Risk Assessment		Exposure Point Concentration **
				(Y/N)?	Reason *	
123PDA	1: 72	19	-	N	4	
24DNT	12: 72	810	3.15	Y		810
26DNT	10: 72	32.5	0.783	Y		32.5
B2EHP	2: 72	1.61	1.56	N	4	
BAANTR	4: 72	0.666	0.173	Y		0.666
BBFANT	2: 72	2.13	2.03	Y		2.13
BGHIPIY	1: 72	1.91	-	Y		1.91
CHRY	8: 72	1	0.08	Y		1
CR	66: 66	109	3.41	Y		109
DEP	37: 72	49.8	0.652	Y		49.8
FANT	20: 72	1.12	0.046	Y		1.12
HG	17: 66	0.716	0.054	Y		0.716
NG	42: 66	1500	0.709	Y		1500
NIT	65: 66	120	1.36	Y		120
NNDMEA	7: 72	0.302	0.022	Y		0.302
NNDNPA	5: 72	0.23	0.096	Y		0.23
NNDPA	58: 72	10000	0.092	Y		10000
PB	66: 66	3500	8.5	Y		3500
PHANTR	14: 72	0.279	0.076	Y		0.279
PYR	8: 72	0.932	0.179	Y		0.932
SO4	17: 66	22.9	6.21	Y		22.9

Footnotes:

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential for human nutrition.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum

Note: Assessment of surface soil contamination (0 to 2 feet) was performed using samples from RPS-91-03 through RPS-91-68.

**Table O-58**  
**Compounds Detected**  
**Rocket Paste Pond Sediment**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	<u>Concentration **</u>
CR	2: 2	45.7	33.8	Y		45.7
DEP	1: 2	2.46	-	Y		2.46
HG	2: 2	0.157	0.08	Y		0.157
NG	1: 2	1.76	-	Y		1.76
NIT	2: 2	2.22	1.96	Y		2.22
NNDPA	2: 2	4.98	0.738	Y		4.98
PB	2: 2	2600	1100	Y		2600
SO4	2: 2	210	150	Y		210

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant.  
                   \* 3 = essential for human nutrition.  
                   \* 4 = frequency of detection less than 5 %.  
                   \*\* 95th percentile or maximum

Note:           Assessment of sediment contamination was performed using samples  
                   from RPS-91-01 and RPS-91-02.

**Table O-59**  
**Compounds Detected**  
**Rocket Paste Pond Surface Water**  
**Units: ug/L**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
AL	2: 2	31400	5410	Y		31400
AS	2: 2	15	8.6	Y		15
BA	2: 2	290	121	Y		290
BE	1: 2	2.17	-	Y		2.17
CA	2: 2	38200	30800	N	3	
CL	2: 2	2730	2700	Y		2730
CR	1: 2	59.5	-	Y		59.5
CU	2: 2	79.1	21.3	Y		79.1
FE	2: 2	31700	7980	N	3	
K	2: 2	44000	43000	N	3	
MG	2: 2	20900	14900	N	3	
MN	2: 2	503	152	Y		503
NA	2: 2	2000	1190	N	3	
NH3N2	2: 2	63.4	33.8	Y		63.4
NI	1: 2	40.7	-	Y		40.7
NIT	1: 2	10.5	-	Y		10.5
PB	2: 2	3100	910	Y		3100
SO4	2: 2	35000	32000	Y		35000
V	2: 2	57.1	22.3	Y		57.1
ZN	2: 2	151	34.9	Y		151

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant.  
                   \* 3 = essential for human nutrition.  
                   \* 4 = frequency of detection less than 5 %  
                   \*\* 95th percentile or maximum

Note:           Assessment of surface water contamination was performed using samples  
                   RPW-91-01 and RPW-91-02.

TABLE O-40

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
ROCKET PASTE AREA  
BADGER ARMY AMMUNITION PLANT

RP ASS 304 25 - Mar - 95

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Medium	mg/kg	USEPA 1991
INGESTION RATE - ADULT	IRa	100	mg/day	USEPA 1991
INGESTION RATE - CHILD	IRc	200	mg/day	USEPA 1991
FRACTION INGESTED	FI	100%		Assumption
CONVERSION FACTOR	CF	0.000001	kg/mg	USEPA 1991
BODY WEIGHT - ADULT	BWa	70	kg	USEPA 1991
BODY WEIGHT - CHILD	BWc	15	kg	USEPA 1991
EXPOSURE FREQUENCY	EF	350	days/year	USEPA 1991
EXPOSURE DURATION - ADULT	EDa	24	years	USEPA 1991
EXPOSURE DURATION - CHILD	EDc	6	years	USEPA 1991
AVERAGING TIME				
CANCER	AT	70	years	USEPA 1989
ADULT - NONCANCER	ATa	24	years	USEPA 1991
CHILD - NONCANCER	ATc	6	years	USEPA 1991
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	USEPA 1989

USEPA 1991. Risk Assessment Guidelines for Superfund  
USEPA 1991. Standard Default Exposure Factors

Note:

For noncarcinogenic effects: AT = ED

CANCER RISK = INTAKE (mg/kg-day) x CANCER SLOPE FACTOR (mg/kg-day)<sup>-1</sup>

HAZARD QUOTIENT = INTAKE (mg/kg-day) / REFERENCE DOSE (mg/kg-day)

INTAKE-ADULT =  $\frac{CS \times IRa \times FI \times CF \times EF \times EDa}{BWa \times ATa \times SAS \text{ days/yr}}$

INTAKE-CHILD =  $\frac{CS \times IRc \times FI \times CF \times EF \times EDc}{BWc \times ATc \times SAS \text{ days/yr}}$

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
ROCKET PASTE AREA  
BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAT	INTAKE (mg/kg-day)		CANCER FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK		TOTAL CANCER RISK
			ADULT	CHILD		ADULT	CHILD	
ACNT	610	1	3.8E-04	8.9E-04	6.8E-01	2.6E-04	6.0E-04	8.6E-04
ACNT	575	1	1.5E-03	3.4E-03	6.8E-01	1.0E-03	2.4E-03	3.5E-03
BAANT	0.666	1	3.1E-07	7.3E-07	7.3E+00	2.3E-06	5.5E-06	7.6E-06
BSFANT	2.13	1	1.0E-06	2.3E-06	7.3E+00	7.3E-06	1.7E-05	2.4E-05
CBRY	1	1	4.7E-07	1.1E-06	7.3E+00	3.4E-06	8.0E-06	1.1E-05
CR	109	1	5.1E-03	1.2E-04	ND			
INDMELA	0.502	1	1.4E-07	3.3E-07	5.1E+01	7.2E-06	1.7E-05	2.4E-05
INDMFA	0.23	1	1.1E-07	2.5E-07	7.0E+00	7.6E-07	1.8E-06	2.5E-06
INDFA	10000	1	4.7E-03	1.1E-02	4.9E-03	2.3E-05	5.4E-05	7.7E-05
FB	3500	1	1.4E-03	3.4E-03	ND			
SUMMARY CANCER RISK								
						3E-04	7E-04	1E-03

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
ROCKET PASTE AREA  
BADGER ARMY AMMUNITION PLANT

## NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAI	INTAKE		REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT		TOTAL HAZARD QUOTIENT
			ADULT (mg/kg-day)	CHILD (mg/kg-day)		ADULT	CHILD	
2MONT	810	1	1.1E-03	1.0E-02	2.0E-03	5.53E-01	5.18E+00	5.73E+00
2MONT	32.5	1	4.5E-05	4.2E-04	ND			
BAAMTR	0.666	1	9.1E-07	8.5E-06	4.0E-02	2.28E-05	2.13E-04	2.36E-04
BBPANT	2.13	1	2.9E-06	2.7E-05	4.0E-02	7.29E-05	6.81E-04	7.54E-04
BCBEPY	1.91	1	2.6E-06	2.4E-05	4.0E-02	6.54E-05	6.11E-04	6.76E-04
CBRY	1	1	1.4E-06	1.3E-05	4.0E-02	3.42E-05	3.20E-04	3.54E-04
CR	109	1	1.5E-04	1.4E-03	5.0E-03	2.99E-02	2.79E-01	3.09E-01
DEP	49.8	1	6.8E-03	6.4E-04	8.0E-01	8.53E-05	7.96E-04	8.81E-04
PANT	1.12	1	1.5E-06	1.4E-05	4.0E-02	3.84E-05	3.58E-04	3.96E-04
EG	0.716	1	9.8E-07	9.2E-06	3.0E-04	3.27E-05	3.05E-02	3.98E-02
NO	1500	1	2.1E-03	1.9E-02	ND			
NET	120	1	1.6E-04	1.5E-03	1.0E-01	1.64E-03	1.53E-02	1.70E-02
NONMZA	0.502	1	4.1E-07	3.9E-06	ND			
NONNFA	0.23	1	3.2E-07	2.9E-06	ND			
NONFA	10000	1	1.4E-02	1.3E-01	ND			
PS	3500	1	4.8E-03	4.5E-02	ND			
PEANTR	0.279	1	3.8E-07	3.6E-06	4.0E-02	9.55E-06	8.92E-05	9.87E-05
PTB	0.932	1	1.3E-06	1.2E-05	3.0E-02	4.26E-05	3.97E-04	4.40E-04
SO4	22.9	1	3.1E-05	2.9E-04	ND			
SUMMARY HAZARD INDEX						0.4	5.5	6.1

TABLE O-61

INCIDENTAL INGESTION AND INHALATION OF SOIL.  
 GROUNDS MAINTENANCE WORKER  
 ROCKET PASTE AREA  
 BADGER ARMY AMMUNITION PLANT

RPASSGJW 06 - Dec - 92

EXPOSURE PARAMETERS

EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Maximum	mg/kg	USEPA, 1991a
INGESTION RATE	IR	100	mg/day	Assumption
FRACTION INGESTED	FI	100%		
CONVERSION FACTOR	CF	0.000001	kg/mg	
BODY WEIGHT	BW	70	kg	USEPA, 1991a
EXPOSURE FREQUENCY	EF	24	days/year	USEPA, 1991a
EXPOSURE DURATION	ED	25	years	USEPA, 1991a
CONCENTRATION AIR PARTICULATES	CAp	Calculated	mg/m <sup>3</sup>	Appendix M
CONCENTRATION AIR VOLATILES	CAv	Calculated	mg/m <sup>3</sup>	USEPA, 1991b
VOLATILIZATION FACTOR	VF	Calculated	m <sup>3</sup> /kg	USEPA, 1991a
PARTICULATE EMISSION FACTOR	PEF	4.63E+09	m <sup>3</sup> /hour	Assumption
INHALATION RATE	IIR	2.5	m <sup>3</sup> /hour	
EXPOSURE TIME	ET	8	hours/day	
AVERAGING TIME	AT			
CANCER				
NONCANCER				
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	USEPA, 1989

$$\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$$

$$\text{HAZARD QUOTIENT}_{\text{Ingestion}} = \text{INTAKE (mg/kg-day)} / \text{REFERENCE DOSE (mg/kg-day)}$$

$$\text{HAZARD QUOTIENT}_{\text{Inhalation}} = \frac{\text{AIR CONCENTRATION (mg/m}^3\text{)}}{\text{REFERENCE CONCENTRATION (mg/m}^3\text{)}}$$

$$\text{INTAKE-INGESTION} = \frac{\text{CS} \times \text{IR} \times \text{RAF} \times \text{FI} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

$$\text{INTAKE-INHALATION} = \frac{(\text{CAp} + \text{CAv}) \times \text{IIR} \times \text{ET} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

$$\text{AIR CONCENTRATION (mg/m}^3\text{)} = \text{CAp} + \text{CAv}$$

$$\text{CAp} = \text{CS} \times \text{I/P EF}$$

$$\text{CAv} = \text{CS} \times \text{U/PF}$$

Note:

For carcinogenic effects: AT = ED

USEPA, 1989. Risk Assessment Guidelines for Superfund - Part A  
 USEPA, 1990. Exposure Factors Handbook  
 USEPA, 1991a. Standard Default Exposure Factors  
 USEPA, 1991b. Risk Assessment Guidelines for Superfund - Part B

TABLE O-61, continued  
INCIDENTAL INGESTION AND INHALATION OF SOIL  
GROUNDS MAINTENANCE WORKER  
ROCKET PASTE AREA  
BADGER ARMY AMMUNITION PLANT

RPASSJW 08-Dec-92

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION BAF	INTAKE		CANCER SLOPE FACTOR-ING. (mg/kg-day) <sup>-1</sup>	CANCER SLOPE FACTOR-ING. (mg/kg-day) <sup>-1</sup>	CANCER RISK		TOTAL CANCER RISK
			INGESTION (mg/kg-day)	INHALATION (mg/kg-day)			INGESTION	INHALATION	
24DNT	810	1	2.7E-05	1.2E-09	ND	6.8E-01	1.8E-05		1.8E-05
26DNT	32.5	1	1.1E-06	4.7E-11	ND	6.8E-01	7.4E-07		7.4E-07
BAANTR	0.666	1	2.2E-06	9.7E-13	6.1E+00	7.3E+00	1.6E-07	5.9E-12	1.6E-07
BEFANT	2.13	1	7.1E-06	3.1E-12	6.1E+00	7.3E+00	5.2E-07	1.9E-11	5.2E-07
CHRY	1	1	3.4E-06	1.4E-12	6.1E+00	7.3E+00	2.4E-07	8.8E-12	2.4E-07
CR	109	1	3.7E-06	1.6E-10	4.1E+01	ND		6.5E-09	
NNDMBA	0.302	1	1.0E-06	4.4E-13	5.1E+01	5.1E+01	5.2E-07	2.2E-11	5.2E-07
NNDMPA	0.23	1	7.7E-09	3.3E-13	ND	7.0E+00	5.4E-08		5.4E-08
NNDPA	10000	1	3.4E-04	1.4E-08	ND	4.9E-03			
PB	3900	1	1.3E-04	5.1E-09	ND	ND			
SUMMARY CANCER RISK									
							2E-05	7E-09	2E-05



TABLE O-61, continued

RPASSOW 06-1 Dec-92

INCIDENTAL INGESTION AND INHALATION OF SOIL.  
 GROUNDS MAINTENANCE WORKER  
 ROCKET PASTE AREA  
 BADGER ARMY AMMUNITION PLANT

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION BAF	INTAKE (mg/kg-day)	AIR CONCENT. (mg/m <sup>3</sup> )	REFERENCE CONCENT. (mg/m <sup>3</sup> )	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT INHALATION	TOTAL HAZARD QUOTIENT
24DNT	810	1	7.6E-05	1.7E-07	ND	2.0E-03	3.80E-02		3.80E-02
24DNT	32.5	1	3.1E-06	7.0E-09	ND	ND			
BAANTR	0.666	1	6.3E-08	1.4E-10	ND	4.0E-02	1.56E-06		1.56E-06
BREANT	2.13	1	2.0E-07	4.6E-10	ND	4.0E-02	5.00E-06		5.00E-06
BGIOPY	1.91	1	1.8E-07	4.1E-10	ND	4.0E-02	4.49E-06		4.49E-06
CBRY	1	1	9.4E-08	2.2E-10	ND	4.0E-02	2.35E-06		2.35E-06
CR	109	1	1.0E-05	2.4E-08	ND	5.0E-03	2.05E-03		2.05E-03
DEP	49.8	1	4.7E-06	1.1E-08	ND	8.0E-01	5.85E-06		5.85E-06
FANT	1.12	1	1.1E-07	2.4E-10	ND	4.0E-02	2.63E-06		2.63E-06
HG	0.716	1	6.7E-08	1.5E-10	3.0E-04	3.0E-04	2.24E-04		2.24E-04
NO	1500	1	1.4E-04	3.2E-07	ND	ND			
NIT	120	1	1.1E-05	2.6E-08	ND	1.0E-01	1.13E-04		1.13E-04
NNDMA	0.302	1	2.8E-08	6.5E-11	ND	ND			
NNDMA	0.23	1	2.2E-08	5.0E-11	ND	ND			
NNDPA	10000	1	9.4E-04	2.2E-06	ND	ND			
PB	3500	1	3.3E-04	7.6E-07	ND	ND			
PIANTR	0.279	1	2.6E-08	6.0E-11	ND	4.0E-02	6.55E-07		6.55E-07
PYR	0.932	1	8.8E-08	2.0E-10	ND	3.0E-02	2.92E-06		2.92E-06
SO4	22.9	1	2.2E-06	4.9E-09	ND	ND			
SUMMARY HAZARD INDEX							0.04	0.00	0.04

TABLE O-62

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SEDIMENT  
 OLDER CHILD (6-16 Years) EXPLORING  
 RAYKIE ARMY AMMUNITION PLANT  
 ROCKY PASTE AREA POND

RPASST

25-MAR-93

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Maximum	mg/kg	USEPA, 1991
INGESTION RATE	IR	100	mg/day	Assumption
FRACTION INGESTED	FI	100%		USEPA, 1992
SOIL ADHERENCE FACTOR	SAF	1	mg/cm <sup>2</sup>	USEPA, 1990
SURFACE AREA EXPOSED	SA	6.150	cm <sup>2</sup> /day	Assumption
CONVERSION FACTOR	CF	0.000001	kg/mg	Assumption
BODY WEIGHT	BW	40	kg	USEPA, 1990
EXPOSURE FREQUENCY	EF	50	days/year	Assumption
EXPOSURE DURATION	ED	11	years	Assumption
AVERAGING TIME	AT	70	years	USEPA, 1989
RELATIVE ABSORPTION FACTOR	RAF	11	years	USEPA, 1989
CANCER NONCANCER INGESTION DERMAL		1	unitless	USEPA, 1989

USEPA, 1989, Risk Assessment Guidance for Superfund

USEPA, 1990, Exposure Factors Handbook

USEPA, 1991, Standard Default Exposure Factors

USEPA, 1992, Dermal Exposure Guidance

Note:

For noncarcinogenic effects: AT = ED

CANCER RISK =  $\text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$ HAZARD QUOTIENT =  $\text{INTAKE (mg/kg-day)} / \text{REFERENCE DOSE (mg/kg-day)}$ INTAKE =  $(\text{INTAKE-INGESTION}) + (\text{INTAKE-DERMAL})$ 

$$\text{INTAKE-INGESTION} = \frac{\text{CS} \times \text{IR} \times \text{RAF} \times \text{FI} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

$$\text{INTAKE-DERMAL} = \frac{\text{CS} \times \text{SA} \times \text{SAF} \times \text{RAF} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

TAB. E. O - 82. continued

RPASST 25-Mar-91

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SEDIMENT  
 OLDER CHILD (6-16 Years) EXPLORING  
 BAUGER ARMY AMMUNITION PLANT  
 ROCKET PASTE AREA POND

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAP	INTAKE INGESTION (mg/kg-day)	DERMAL RAP	INTAKE DERMAL (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK DERMAL	TOTAL CANCER RISK
CR	45.7	1	2.7E-06	No values available for Quantitative Analysis	ND	ND	1.3E-09		1.3E-09
MEPPA	4.98	1	2.7E-07		4.9E-03	ND			
PS	2600	1	1.4E-04		ND				
SUMMARY CANCER RISK							1E-09	0E+00	1E-09

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SEDIMENT  
 OLDER CHILD (6-16 Years) EXPLORING  
 RADGEE ARMY AMMUNITION PLANT  
 RUCKET PASTE AREA POND

## NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAP	INTAKE INGESTION (mg/kg-day)	DERMAL RAP	INTAKE DERMAL (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT DERMAL	TOTAL HAZARD QUOTIENT
CR	45.7	1	1.6E-05	No values available for Quantitative Analysis		5.0E-03	3.13E-03		3.13E-03
DEP	2.46	1	8.4E-07			8.0E-01	1.05E-06		1.05E-06
INO	0.157	1	5.4E-08			3.0E-04	1.79E-04		1.79E-04
NO	1.76	1	6.0E-07			ND			
NT	2.22	1	7.6E-07			1.0E-01	7.60E-06		7.60E-06
NDPA	4.96	1	1.7E-06			ND			
PB	2600	1	8.9E-04			ND			
SO4	210	1	7.2E-05			ND			
SUMMARY HAZARD INDEX							0.003	0.000	0.003

INGESTION OF AND DERMAL CONTACT WITH SURFACE WATER  
 OLDER CHILD (6 - 10) EXPLORING  
 ROCKET PASTE AREA POND  
 BADGER ARMY AMMUNITION PLANT

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION WATER	CW	Medium	mg/liter	USEPA 1991
INGESTION RATE	IR	0.05	liter/hour	USEPA 1990
SURFACE AREA EXPOSED	SA	13,000	cm <sup>2</sup>	USEPA 1990
BODY WEIGHT	BW	40	kg	USEPA 1990
CONVERSION FACTOR	CF	0.001	liter/cm <sup>2</sup>	USEPA 1999
EXPOSURE TIME	ET	2.6	hour/day	USEPA 1999
EXPOSURE FREQUENCY	EF	7	days/year	USEPA 1999
EXPOSURE DURATION	ED	11	years	Assumption
AVERAGING TIME	AT	70	years	USEPA 1991
CANCER	AT	11	years	USEPA 1991
NONCANCER	RAF	1	unitless	USEPA 1999
RELATIVE ABSORPTION FACTOR	RAF	0.00153	cm <sup>2</sup> /hour	USEPA 1992
PERMEABILITY CONSTANT	PC			

USEPA, 1999, Risk Assessment Guidance for Superfund

USEPA, 1990, Exposure Factors Handbook

USEPA, 1991, Standard Default Exposure Factors

USEPA, 1992, Dermal Exposure Factors

Note:

Per concentration specific: AT = ED

$$\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$$

$$\text{HAZARD QUOTIENT} = \text{INTAKE (mg/kg-day)} / \text{REFERENCE DOSE (mg/kg-day)}$$

$$\text{INTAKE} = (\text{INTAKE} - \text{INGESTION}) + (\text{INTAKE} - \text{DERMAL})$$

$$\text{INTAKE} - \text{INGESTION} = \frac{\text{CWF} \times \text{IR} \times \text{SA} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

$$\text{INTAKE} - \text{DERMAL} = \frac{\text{CWF} \times \text{SA} \times \text{PC} \times \text{ET} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

INGESTION OF AND DERMAL CONTACT WITH SURFACE WATER  
 OLDER CHILD (6 - 16) EXPLORING  
 ROCKET PASTE AREA POND  
 BADGER ARMY AMMUNITION PLANT

RPASWT 08-Dec-92

CARCINOGENIC EFFECTS

COMPOUND	WATER CONCENTRATION (mg/l)	INGESTION RBP	INTAKE INGESTION (mg/kg-day)	PERMEABILITY CONSTANT (cm/hr)	INTAKE DERMAL (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK DERMAL	TOTAL CANCER RISK
AS	0.015	1	5.7E-08	1.55E-05	5.9E-08	1.8E+00	1.0E-07	1.1E-07	2.1E-07
BB	0.00217	1	8.2E-09	1.55E-05	8.6E-09	4.3E+00	3.5E-08	3.7E-08	7.2E-08
CR	0.0595	1	2.2E-07	1.55E-05	2.3E-07	ND			
PS	3.1	1	1.2E-05	1.55E-05	1.2E-05	ND			
SUMMARY CANCER RISK									
							1B-07	1B-07	3E-07

## INGESTION OF AND DERMAL CONTACT WITH SURFACE WATER

OLDER CHILD (6 - 16) EXPLORING

ROCKET PASTE AREA POND

BADGER ARMY AMMUNITION PLANT

## NONCARCINOGENIC EFFECTS

COMPOUND	WATER CONCENTRATION (mg/l)	INGESTION EAF	INTAKE INGESTION (mg/kg-day)	PERMEABILITY CONSTANT (PC) (cm/hr)	INTAKE DERMAL (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT DERMAL	TOTAL HAZARD QUOTIENT
AL	31.4	1	7.9E-04	1.55E-03	7.9E-04	ND	1.2E-03	1.3E-03	2.5E-03
AS	0.015	1	3.6E-07	1.55E-03	3.6E-07	3.0E-04	9.9E-03	1.0E-04	2.0E-04
BA	0.29	1	7.0E-06	1.55E-03	7.0E-06	7.0E-02	1.0E-03	1.1E-05	2.1E-05
BE	0.00217	1	5.2E-08	1.55E-03	5.2E-08	5.0E-03	ND	ND	ND
CL	2.73	1	6.3E-05	1.55E-03	6.3E-05	5.0E-03	2.0E-04	3.0E-04	5.0E-04
CR	0.0395	1	1.4E-06	1.55E-03	1.4E-06	ND	ND	ND	ND
CU	0.0791	1	1.9E-06	1.55E-03	1.9E-06	ND	1.2E-04	1.3E-04	2.5E-04
MO	0.503	1	1.2E-05	1.55E-03	1.2E-05	1.0E-01	4.9E-03	5.1E-03	1.0E-04
NI	0.0407	1	9.8E-07	1.55E-03	1.0E-06	2.0E-02	2.5E-06	2.6E-06	5.2E-06
NI	0.0105	1	2.5E-07	1.55E-03	2.6E-07	1.0E-01	ND	ND	ND
PD	3.1	1	7.4E-05	1.55E-03	7.4E-05	ND	2.0E-04	2.0E-04	4.0E-04
SO4	35	1	8.4E-04	1.55E-03	8.4E-04	ND	1.8E-05	1.9E-05	3.7E-05
V	0.0371	1	1.4E-06	1.55E-03	1.4E-06	7.0E-05	ND	ND	ND
ZN	0.151	1	3.6E-06	1.55E-03	3.6E-06	2.0E-01	ND	ND	ND
NEUPT	0.0034	1	1.5E-06	1.55E-03	1.6E-06	ND	ND	ND	ND
SUMMARY HAZARD INDEX									0.004

**Table O-64**  
**Compounds Detected**  
**Nitroglycerine Pond Surface Soil**  
**Units:ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	<u>Concentration **</u>
CR	2:2	39.5	32.2	N	1	
HG	1:2	2.4	-	Y		2.4
NG	2:2	15.8	9.39	Y		15.8
NH3	2:2	17.7	4.47	Y		17.7
PB	2:2	10000	2000	Y		10000

Footnotes: \* 1 = within background range.  
\* 2 = laboratory or sampling contaminant.  
\* 3 = essential for human nutrition.  
\* 4 = frequency of detection less than 5 %.  
\*\* 95th percentile or maximum

Note: Assessment of surface soil contamination was performed using samples from NPS-91-09 and NPS-91-10.



**Table O-65**  
**Compounds Detected**  
**Nitroglycerine Pond Sediment**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
CR	8 : 8	40.5	4.9	Y		40.5
HG	8 : 8	20	0.159	Y		20
NH3	8 : 8	72.5	2.28	Y		72.5
PB	8 : 8	410	32	Y		410

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant.  
                   \* 3 = essential for human nutrition.  
                   \* 4 = frequency of detection less than 5 %.  
                   \*\* 95th percentile or maximum

Note:           Assessment of sediment contamination was performed using samples  
                   from NPS-91-01 through NPS-91-08.

**Table O-66**  
**Compounds Detected**  
**Nitroglycerine Pond Surface Water**  
**Units: ug/L**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	<u>Concentration **</u>
AL	2 : 2	3020	2140	Y		3020
AS	2 : 2	5.43	4.98	Y		5.43
BA	2 : 2	63.1	47.3	Y		63.1
CA	2 : 2	15200	11700	N	3	
CL	2 : 2	1930	1680	Y		1930
FE	2 : 2	3970	2920	N	3	
HG	2 : 2	0.325	0.324	Y		0.325
K	2 : 2	15000	12800	N	3	
MG	2 : 2	5880	5340	N	3	
MN	2 : 2	207	81.7	Y		207
NA	2 : 2	8320	7790	N	3	
NH3N2	2 : 2	147	63.4	Y		147
PB	2 : 2	45.9	41.2	Y		45.9
SO4	2 : 2	4470	4070	Y		4470
V	2 : 2	8.37	6.62	Y		8.37

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant.  
                   \* 3 = essential for human nutrition.  
                   \* 4 = frequency of detection less than 5 %.  
                   \*\* 95th percentile or maximum

Note:           Assessment of surface water contamination was performed using samples  
                   NPW-91-01 and NPW-91-02.

TABLE O-67

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
NITROGLYCERINE POND  
BADGER ARMY AMMUNITION PLANT

NPDES 301  
23 - Mar - 93

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Medium	mg/kg	USEPA 1991
INGESTION RATE - ADULT	IRa	100	mg/day	USEPA 1991
INGESTION RATE - CHILD	IRc	200	mg/day	USEPA 1991
FRACTION INGESTED	FI	100%		Assumption
CONVERSION FACTOR	CF	0.000001	kg/mg	USEPA 1991
BODY WEIGHT - ADULT	BWa	70	kg	USEPA 1991
BODY WEIGHT - CHILD	BWc	15	kg	USEPA 1991
EXPOSURE FREQUENCY	EF	350	days/year	USEPA 1991
EXPOSURE DURATION - ADULT	EDa	24	years	USEPA 1991
EXPOSURE DURATION - CHILD	EDc	6	years	USEPA 1991
AVERAGING TIME	AT	70	years	USEPA 1989
ADULT - NONCANCER	ATa	24	years	USEPA 1991
CHILD - NONCANCER	ATc	6	years	USEPA 1991
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	USEPA 1989
<p>CANCER RISK = INTAKE (mg/kg-day) x CANCER SLOPE FACTOR (mg/kg-day)<sup>-1</sup></p> <p>HAZARD QUOTIENT = INTAKE (mg/kg-day) / REFERENCE DOSE (mg/kg-day)</p> <p>INTAKE-ADULT = <math>\frac{CS \times IRa \times RAF \times FI \times CF \times EF \times EDa}{BWa \times ATa \times 365 \text{ days/yr}}</math></p> <p>INTAKE-CHILD = <math>\frac{CS \times IRc \times RAF \times FI \times CF \times EF \times EDc}{BWc \times ATc \times 365 \text{ days/yr}}</math></p> <p>Note: For noncarcinogenic effects: AT = ED</p>				
<p>USEPA, 1989, Risk Assessment Guidance for Superfund USEPA, 1990, Exposure Factors Handbook USEPA, 1991, Standard Default Exposure Factors</p>				

TABLE O-47, continued

INCIDENTAL INGESTION OF SURFACE SOIL.  
RESIDENTIAL - ADULT AND CHILD.  
NITROGLYCERINE POND  
BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAF	INTAKE ADULT (mg/kg-dm)	INTAKE CHILD (mg/kg-dm)	CANCER SLOPE FACTOR (mg/kg-dm) <sup>-1</sup>	CANCER RISK ADULT	CANCER RISK CHILD	TOTAL CANCER RISK
PS	1000	1	4.7E-03	1.1E-02	ND			
SUMMARY CANCER RISK								
						0E+00	0E+00	0E+00

TABLE O-67, continued

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
NITROGLYCERINE POND  
BADGER ARMY AMMUNITION PLANT

## NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RATE	INTAKE		REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT		TOTAL HAZARD QUOTIENT
			ADULT (mg/kg-day)	CHILD (mg/kg-day)		ADULT	CHILD	
BO	24	1	3.5E-06	3.1E-03	3.0E-04	0.0110	0.1023	0.1132
MO	158	1	2.2E-05	2.0E-04	ND			
NES	177	1	2.4E-05	2.3E-04	ND			
PS	1000	1	1.4E-02	1.3E-01	ND			
SUMMARY HAZARD INDEX								0.1132

INCIDENTAL INGESTION AND INHALATION OF SOIL  
 GROUNDS MAINTENANCE WORKER  
 NITROGLYCERINE POND  
 BADGER ARMY AMMUNITION PLANT

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Medium	mg/kg	USEPA, 1991a
INGESTION RATE	IR	100	mg/day	Assumption
FRACTION INGESTED	FI	100%		
CONVERSION FACTOR	CF	0.00001	kg/mg	
BODY WEIGHT	BW	70	kg	USEPA, 1991a
EXPOSURE FREQUENCY	EF	24	days/year	USEPA, 1991a
EXPOSURE DURATION	ED	25	years	USEPA, 1991a
CONCENTRATION AIR PARTICULATES	CAp	Calculated	mg/m <sup>3</sup>	Appendix M
CONCENTRATION AIR VOLATILES	CAv	Calculated	mg/m <sup>3</sup>	USEPA, 1991b
VOLATILIZATION FACTOR	VF	Calculated	m <sup>3</sup> /kg	USEPA, 1991a
PARTICULATE EMISSION FACTOR	PEF	4.63E+09	m <sup>3</sup> /hour	Assumption
INHALATION RATE	IR	2.5	m <sup>3</sup> /hour	
EXPOSURE TIME	ET	8	hour/day	
AVERAGING TIME	AT	70	years	USEPA, 1989
	AT	25	years	USEPA, 1991a
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	USEPA, 1989

USEPA, 1989. Risk Assessment Guidelines for Superfund - Part A

USEPA, 1990. Exposure Factors Handbook

USEPA, 1991b. Risk Assessment Guidelines for Superfund - Part B

USEPA, 1991a. Standard Default Exposure Factors

$$\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$$

$$\text{HAZARD QUOTIENT}_{\text{Ingestion}} = \text{INTAKE (mg/kg-day)} / \text{REFERENCE DOSE (mg/kg-day)}$$

$$\text{HAZARD QUOTIENT}_{\text{Inhalation}} = \frac{\text{AIR CONCENTRATION (mg/m}^3\text{)}}{\text{REFERENCE CONCENTRATION (mg/m}^3\text{)}}$$

$$\text{INTAKE - INGESTION} = \frac{\text{CS} \times \text{IR} \times \text{RAF} \times \text{FI} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/year}}$$

$$\text{INTAKE - INHALATION} = \frac{(\text{CAp} + \text{CAv}) \times \text{IR} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/year}}$$

$$\text{AIR CONCENTRATION (mg/m}^3\text{)} = \text{CAp} + \text{CAv}$$

$$\text{CAp} = \text{CS} \times 1/\text{VF}$$

$$\text{CAv} = \text{CS} \times 1/\text{VF}$$

Note:

For noncarcinogenic effects: AT = ED

TABLE O-68, continued

INCIDENTAL INGESTION AND INHALATION OF SOIL  
 GROUNDS MAINTENANCE WORKER  
 NITROGLYCERINE POND  
 BADGER ARMY AMMUNITION PLANT

NPSS/JW 06 - Dec - 92

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAP	INGESTION (mg/kg-day)	INTAKE INHALATION (mg/kg-day)	CANCER SLOPE FACTOR - INH. (mg/kg-day) <sup>-1</sup>	CANCER SLOPE FACTOR - ING. (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK INHALATION	TOTAL CANCER RISK
FS	1000	1	3.4E-04	1.4E-08	ND	ND			
SUMMARY CANCER RISK									0E+00
									0E+00

INCIDENTAL INGESTION AND INHALATION OF SOIL  
 GROUNDS MAINTENANCE WORKER  
 NITROGLYCERINE POND  
 BADGER ARMY AMMUNITION PLANT

## NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION BAF	INTAKE INGESTION (mg/kg-day)	AIR CONCENT. (mg/m <sup>3</sup> )	REFERENCE CONCENT. (mg/m <sup>3</sup> )	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT INHALATION	TOTAL HAZARD QUOTIENT
HG	2.4	1	2.3E-07	5.2E-10	3.0E-04	3.0E-04	7.51E-04	1.73E-06	7.53E-04
NO	15.8	1	1.5E-06	3.4E-09	ND	ND			
NIS	17.7	1	1.7E-06	3.8E-09	ND	ND			
PB	10000	1	9.4E-04	2.2E-06	ND	ND			
SUMMARY HAZARD INDEX							0.0008	0.0000	0.0008



TABLE O-69

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SEDIMENT  
 OLDER CHILD (6-16 Years) EXPLORING  
 NITROGLYCERINE POND  
 BADGER ARMY AMMUNITION PLANT

NPSST

25-Mar-93

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Maximum	mg/kg	USEPA, 1991
INGESTION RATE	IR	100	mg/day	Assumption
FLACTION INGESTED	FI	100%		USEPA, 1992
SOIL ADHERENCE FACTOR	SAF	1	mg/cm <sup>2</sup>	USEPA, 1990
SURFACE AREA EXPOSED	SA	6,150	cm <sup>2</sup> /day	USEPA, 1990
CONVERSION FACTOR	CF	0.000001	kg/mg	USEPA, 1990
BODY WEIGHT	BW	40	kg	Assumption
EXPOSURE FREQUENCY	EF	50	days/year	Assumption
EXPOSURE DURATION	ED	11	years	USEPA, 1989
AVERAGING TIME	AT	70	years	USEPA, 1989
RELATIVE ABSORPTION FACTOR	RAF	11	years	USEPA, 1989
CANCER				
NONCANCER				
INGESTION				
DERMAL				

USEPA, 1989, Risk Assessment Guidelines for Superfund

USEPA, 1990, Exposure Factors Handbook

USEPA, 1991, Standard Default Exposure Factors

USEPA, 1992, Dermal Exposure Guidelines

Note:

For noncarcinogenic effects: AT = ED

CANCER RISK = INTAKE (mg/kg-day) x CANCER SLOPE FACTOR (mg/kg-day)<sup>-1</sup>

HAZARD QUOTIENT = INTAKE (mg/kg-day) / REFERENCE DOSE (mg/kg-day)

INTAKE = (INTAKE-INGESTION) + (INTAKE-DERMAL)

INTAKE-INGESTION =  $\frac{CS \times IR \times SAF \times FI \times CF \times EF \times ED}{BW \times AT \times 365 \text{ days/yr}}$ INTAKE-DERMAL =  $\frac{CS \times SA \times SAF \times RAF \times CF \times EF \times ED}{BW \times AT \times 365 \text{ days/yr}}$

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SEDIMENT  
 OLDER CHILD (6-16 Years) EXPLORING  
 NITROGLYCERINE POND  
 RADDER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RPF	INGESTION (mg/kg-day)	DERMAL RPF	DERMAL INTAKE (mg/kg-day)	CANCER FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK DERMAL	TOTAL CANCER RISK
CR	40.5	1	2.2E-06	No values available for Quantitative Analysis		ND			
PS	410	1	2.1E-05			ND			
SUMMARY CANCER RISK									0E+00
									0E+00

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SEDIMENT  
 OLDER CHILD (6-16 Years) EXPLORING  
 NITROGLYCERINE POND  
 BADGER ARMY AMMUNITION PLANT

## NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAP	INTAKE INGESTION (mg/kg-day)	DERMAL RAP	INTAKE DERMAL (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT DERMAL	TOTAL HAZARD QUOTIENT
CR	40.5	1	1.4E-05	No values available for Quantitative Analysis		5.0E-05	0.0028		0.0028
HC	20	1	6.8E-06			3.0E-04	0.0226		0.0226
MTS	72.5	1	2.5E-05			ND			
PB	410	1	1.4E-04			ND			
SUMMARY HAZARD INDEX							0.0256	0.0000	0.0256

TABLE O-70

INGESTION OF AND DERMAL CONTACT WITH SURFACE WATER  
 OLDER CHILD (6 - 16) EXPLORING  
 NITROGLYCERINE POND  
 BADGER ARMY AMMUNITION PLANT

NFSWT

06-Dec-92

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION WATER	CW	Median	mg/liter	USEPA 1991
INGESTION RATE	IR	0.05	liter/hour	USEPA 1990
SURFACE AREA EXPOSED	SA	13,000	cm <sup>2</sup>	USEPA 1990
BODY WEIGHT	BW	40	kg	USEPA 1990
CONVERSION FACTOR	CF	0.001	liter/cm <sup>2</sup> s	USEPA 1999
EXPOSURE TIME	ET	2.6	hour/day	USEPA 1999
EXPOSURE FREQUENCY	EF	7	day/year	Assumption
EXPOSURE DURATION	ED	11	years	USEPA 1991
AVERAGING TIME				USEPA 1991
				USEPA 1999
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	USEPA 1992
PERMEABILITY CONSTANT	PC	0.0055	cm/hour	
CANCER NONCANCER USEPA, 1988. Risk Assessment Guidance for Superfund USEPA, 1990. Exposure Factors Handbook USEPA, 1991. Standard Default Exposure Factors USEPA, 1992. Dermal Exposure Guidance				

$$\text{CANCER RISK} = \text{INTAKE} (\text{mg/kg-day}) \times \text{CANCER SLOPE FACTOR} (\text{mg/kg-day})^{-1}$$

$$\text{HAZARD QUOTIENT} = \text{INTAKE} (\text{mg/kg-day}) / \text{REFERENCE DOSE} (\text{mg/kg-day})$$

$$\text{INTAKE} = (\text{INTAKE-INGESTION}) + (\text{INTAKE-DERMAL})$$

$$\text{INTAKE-INGESTION} = \frac{\text{CW} \times \text{IR} \times \text{SA} \times \text{CF} \times \text{ET} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ day/yr}}$$

$$\text{INTAKE-DERMAL} = \frac{\text{CW} \times \text{SA} \times \text{PC} \times \text{CF} \times \text{ET} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ day/yr}}$$

Note:

For noncarcinogenic effects: AT = ED

TABLE O-70, continued

INGESTION OF AND DERMAL CONTACT WITH SURFACE WATER  
 OLDER CHILD (6 - 16) EXPLORING  
 NITROGLYCERINE POND  
 BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	WATER CONCENTRATION (mg/l)	INGESTION RBP	INGESTION (mg/kg-day)	PERMEABILITY CONSTANT (cm/hr)	INTAKE DERMAL (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK DERMAL	TOTAL CANCER RISK
AS	0.00543	1	2.0E-08	0.00155	2.1E-08	1.8E+00	3.7E-08	3.9E-08	7.5E-08
PB	0.0459	1	1.7E-07	0.00155	1.8E-07	ND			
SUMMARY CANCER RISK									4E-08
									8E-08

TABLE O-70. continued

INGESTION OF AND DERMAL CONTACT WITH SURFACE WATER  
OLDER CHILD (6 - 16) EXPLORING  
NITROGLYCERINE POND  
BADGER ARMY AMMUNITION PLANT

NPSWT 08 - Dec - 92

NONCARCINOGENIC EFFECTS

COMPOUND	WATER CONCENTRATION (mg/l)	INGESTION RPF	INTAKE INGESTION (mg/kg-day)	PERMEABILITY CONSTANT (PC) (cm/hr)	INTAKE DERMAL (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT DERMAL	TOTAL HAZARD QUOTIENT
AL	3.02	1	7.2E-05	0.00155	7.6E-05	ND	4.3E-04	4.5E-04	8.9E-04
AS	0.00543	1	1.3E-07	0.00155	1.4E-07	3.0E-04	2.2E-05	2.3E-05	4.4E-05
BA	0.0631	1	1.5E-06	0.00155	1.6E-06	7.0E-02	ND	ND	ND
CL	1.93	1	4.6E-05	0.00155	4.8E-05	ND	2.6E-05	2.7E-05	5.3E-05
HG	0.000325	1	7.8E-09	0.00155	8.2E-09	3.0E-04	5.0E-05	5.2E-05	1.0E-04
MN	0.207	1	5.0E-06	0.00155	5.2E-06	1.0E-01	ND	ND	ND
PB	0.0459	1	1.1E-06	0.00155	1.2E-06	ND	2.9E-05	3.0E-05	5.9E-05
SO4	4.47	1	1.1E-04	0.00155	1.1E-04	ND	ND	ND	ND
V	0.00837	1	2.0E-07	0.00155	2.1E-07	7.0E-03	ND	ND	ND
NITRO2	0.147	1	3.5E-06	0.00155	3.7E-06	ND	ND	ND	ND
SUMMARY HAZARD INDEX							0.0006	0.0006	0.0011

**Table O-71**  
**Compounds Detected**  
**Oleum Plant Surface Soil (0-2')**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
CR	1: 9	14.4	-	N	1	
FE	1: 9	16	-	N	1	
PB	1: 9	6.82	-	N	1	
NIT	3: 3	3.46	1.68	Y		3.46
SO4	3: 9	8500	1000	Y		8500

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant.  
                   \* 3 = essential for human nutrition.  
                   \* 4 = frequency of detection less than 5 %.  
                   \*\* 95th percentile or maximum

Note:           Assessment of surface soil contamination (0 to 2 feet) was performed  
                   using samples from borings OPB-91-01 and OPB-91-06 through OPB-91-13.

**Table O-72**  
**Compounds Detected**  
**Oleum Plant and Pond Subsurface Soil (2'-12')**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	<u>Concentration **</u>
CR	14 : 14	30.3	2.05	Y		30.3
FE	8 : 8	43600	11200	N	3	
HG	1 : 14	0.115	-	Y		0.115
NI	6 : 6	23.1	4.21	Y		23.1
NTT	11 : 14	6.19	1.13	Y		6.19
PB	14 : 14	18	4.76	N	1	
SO4	14 : 14	14000	7.11	Y		14000

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant.  
                   \* 3 = essential for human nutrition.  
                   \* 4 = frequency of detection less than 5 %.  
                   \*\* 95th percentile or maximum

Note:           Assessment of subsurface soil contamination (2 to 12 feet) was performed using samples from borings OPB-91-01 through OPB-91-05.



**Table O-73**  
**Compounds Detected**  
**Oleum Pond Sediment**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
CA	4 : 4	36900	4380	N	3	
NA	3 : 4	120	67.2	N	3	
NIT	4 : 4	50	14	Y		50
SO4	4 : 4	590	160	Y		590

Footnotes:

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential for human nutrition.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum

Note: Assessment of sediment contamination was performed using samples OPS-91-01 through OPS-91-04.

TABLE O-74

INCIDENTAL INGESTION OF SURFACE SOIL.  
RESIDENTIAL - ADULT AND CHILD  
OLEUM PLANT AND POND  
BADGER ARMY AMMUNITION PLANT

CUPSSS-001

75-Mar-93

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Medium	mg/kg	USEPA, 1991
INGESTION RATE - ADULT	IRa	100	mg/day	USEPA, 1991
INGESTION RATE - CHILD	IRc	200	mg/day	USEPA, 1991
FRACTION INGESTED	FI	100%		Assumption
CONVERSION FACTOR	CF	0.000001	kg/mg	USEPA, 1991
BODY WEIGHT - ADULT	BWa	70	kg	USEPA, 1991
BODY WEIGHT - CHILD	BWc	15	kg	USEPA, 1991
EXPOSURE FREQUENCY	EF	350	days/year	USEPA, 1991
EXPOSURE DURATION - ADULT	EDa	24	years	USEPA, 1991
EXPOSURE DURATION - CHILD	EDc	6	years	USEPA, 1991
AVERAGING TIME				
CANCER	AT	70	years	USEPA, 1989
ADULT - NONCANCER	ATa	24	years	USEPA, 1991
CHILD - NONCANCER	ATc	6	years	USEPA, 1991
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	USEPA, 1989

USEPA, 1989. Risk Assessment Guidelines for Superfund  
USEPA, 1991. Standard Default Exposure Factors

Note:

For noncarcinogenic effects: AT = ED

CANCER RISK = INTAKE (mg/kg-day) x CANCER SLOPE FACTOR (mg/kg-day)<sup>-1</sup>

HAZARD QUOTIENT = INTAKE (mg/kg-day) / REFERENCE DOSE (mg/kg-day)

INTAKE-ADULT =

$$CS \times IRa \times FI \times CF \times EF \times EDa \\ BWa \times ATa \times 365 \text{ days/yr}$$

INTAKE-CHILD =

$$CS \times IRc \times FI \times CF \times EF \times EDc \\ BWc \times ATc \times 365 \text{ days/yr}$$

TABLE O-74, continued

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
OLEUM PLANT AND POND  
BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAT	INTAKE ADULT (mg/kg-day)	INTAKE CHILD (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK		CANCER RISK	
						ADULT	CHILD	ADULT	CHILD
No carcinogenic compounds									
SUMMARY CANCER RISK						0E+00	0E+00	0E+00	0E+00

TABLE O-74, continued

INCIDENTAL INGESTION OF SURFACE SOIL.  
RESIDENTIAL - ADULT AND CHILD  
OILFUM PLANT AND POND  
BADGER ARMY AMMUNITION PLANT

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAI	INTAKE		INTAKE		REFERENCE DOSE (mg/kg-dm)	HAZARD QUOTIENT		HAZARD QUOTIENT		TOTAL HAZARD QUOTIENT
			ADULT (mg/kg-dm)	CHILD (mg/kg-dm)	ADULT (mg/kg-dm)	CHILD (mg/kg-dm)		ADULT	CHILD	ADULT	CHILD	
MT	3.46	1	4.7E-06	4.4E-03	1.0E-01	ND	1.0E-01	0.000	0.004	0.000	0.004	0.004
904	8500	1	1.2E-02	1.1E-01	1.1E-01	ND	1.1E-01	0.000	0.000	0.000	0.000	0.000
SUMMARY HAZARD INDEX												0.0004
												0.0005

**TABLE O-73**  
**INCIDENTAL INGESTION AND INHALATION OF SOIL**  
**GROUND MAINTENANCE WORKER**  
**OLEUM PLANT AND POND**  
**BADGER ARMY AMMUNITION PLANT**

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Medium	mg/kg	USEPA, 1991a
INGESTION RATE	IR	100	mg/day	Assumption
FRACTION INGESTED	FI	100%		
CONVERSION FACTOR	CF	0.000001	kg/mg	
BODY WEIGHT	BW	70	kg	USEPA, 1991a
EXPOSURE FREQUENCY	EF	24	days/year	USEPA, 1991a
EXPOSURE DURATION	ED	25	years	USEPA, 1991a
CONCENTRATION AIR PARTICULATES	CAp	Calculated	mg/m <sup>3</sup>	Appendix M
CONCENTRATION AIR VOLATILES	CAv	Calculated	mg/m <sup>3</sup>	USEPA, 1991b
VOLATILIZATION FACTOR	VF	Calculated	m <sup>3</sup> /kg	USEPA, 1991a
PARTICULATE EMISSION FACTOR	PEF	4.63E+09	m <sup>3</sup> /kg	Assumption
INHALATION RATE	IRr	2.5	m <sup>3</sup> /hour	
EXPOSURE TIME	ET	6	hours/day	
AVERAGING TIME				
RELATIVE ABSORPTION FACTOR				
CANCER	AT	70	years	USEPA, 1989
NONCANCER	AT	25	years	USEPA, 1991a
	RAF	1	unitless	USEPA, 1989

CANCER RISK = INTAKE (mg/kg-day)  $\pm$  CANCER SLOPE FACTOR (mg/kg-day)<sup>-1</sup>

HAZARD QUOTIENT<sub>ingestion</sub> = INTAKE (mg/kg-day) / REFERENCE DOSE (mg/kg-day)

HAZARD QUOTIENT<sub>inhalation</sub> =  $\frac{\text{AIR CONCENTRATION (mg/m}^3\text{)}}{\text{REFERENCE CONCENTRATION (mg/m}^3\text{)}}$

INTAKE - INGESTION =  $\frac{\text{CS} \pm \text{IR} \pm \text{RAF} \pm \text{FI} \pm \text{CF} \pm \text{EF} \pm \text{ED}}{\text{BW} \pm \text{AT} \pm 365 \text{ days/yr}}$

INTAKE - INHALATION =  $\frac{(\text{CAp} + \text{CAv}) \pm \text{IRr} \pm \text{ET} \pm \text{VF} \pm \text{ED}}{\text{BW} \pm \text{AT} \pm 365 \text{ days/yr}}$

AIR CONCENTRATION (mg/m<sup>3</sup>) = CAp + CAv

CAp = CS  $\pm$  1/EF

CAv = CS  $\pm$  UVF

Note:

For cancer diopathic effects: AT = ED

USEPA, 1989: Risk Assessment Guidelines for Superfund - Part A

USEPA, 1990: Exposure Factors Handbook

USEPA, 1991a: Standard Default Exposure Factors

USEPA, 1991b: Risk Assessment Guidelines for Superfund - Part B

USEPA, 1989: Risk Assessment Guidelines for Superfund - Part A

TABLE O-75, continued  
INCIDENTAL INGESTION AND INHALATION OF SOIL,  
GROUNDS MAINTENANCE WORKER  
OLEUM PLANT AND POND  
BADGER ARMY AMMUNITION PLANT

OPTSSGW 08-128-92

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RBP	INTAKE INGESTION (mg/kg-day)	INTAKE INHALATION (mg/kg-day)	CANCER SLOPE FACTOR-INEL (mg/kg-day) <sup>-1</sup>	CANCER SLOPE FACTOR-ING. (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK INHALATION	TOTAL CANCER RISK
No carcinogenic compounds									
SUMMARY CANCER RISK									0E+00

TABLE O-75, continued

INCIDENTAL INGESTION AND INHALATION OF SOIL,  
 GROUNDS MAINTENANCE WORKER  
 OLEUM PLANT AND POND  
 BADGER ARMY AMMUNITION PLANT

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08 - Dec - 92

## NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAV	INTAKE INGESTION (mg/kg-day)	AIR CONCENT. (mg/m <sup>3</sup> )	REFERENCE CONCENT. (mg/m <sup>3</sup> )	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT INHALATION	TOTAL HAZARD QUOTIENT
NTT	3.46	1	3.3E-07	7.5E-10	ND	1.0E-01	3.25E-06		3.25E-06
SO4	8500	1	8.0E-04	1.8E-06	ND	ND			
SUMMARY HAZARD INDEX							0.0000	0.0000	3E-06

TABLE 0-76

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (2 - 12 feet)  
CONSTRUCTION WORKER  
OLEUM PLANT AND POND  
BADGER ARMY AMMUNITION PLANT

OPPSR-CW

08-Dec-92

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Maximum	mg/kg	USEPA, 1991
INGESTION RATE	IR	480	mg/day	Assumption
FRACTION INGESTED	FI	100%		USEPA, 1992
SOIL ADHERENCE FACTOR	SAF	1	mg/cm <sup>2</sup>	USEPA, 1990
SURFACE AREA EXPOSED	SA	2,100	cm <sup>2</sup> /day	USEPA, 1990
CONVERSION FACTOR	CF	0.000001	kg/mg	Assumption
BODY WEIGHT	BW	70	kg	USEPA, 1991
EXPOSURE FREQUENCY	EF	20	days/year	Assumption
EXPOSURE DURATION	ED	1	years	Assumption
AVERAGING TIME	AT	70	years	USEPA, 1989
CANCER	AT	0.0547945005	years	Assumption
NONCANCER	AT		years	Assumption
RELATIVE ABSORPTION FACTOR	RAF		unitless	USEPA, 1989
INGESTION				
DERMAL				

USEPA, 1999, Risk Assessment Guidelines for Superfund

USEPA, 1990, Exposure Factors Handbook

USEPA, 1991, Standard Default Exposure Factors

USEPA, 1992, Dermal Absorption Guidelines

CANCER RISK = INTAKE (mg/kg-day) x CANCER SLOPE FACTOR (mg/kg-day)<sup>-1</sup>

HAZARD QUOTIENT = INTAKE (mg/kg-day) / REFERENCE DOSE (mg/kg-day)

INTAKE = (INTAKE-INGESTION) + (INTAKE-DERMAL)

$$\text{INTAKE-INGESTION} = \frac{CS \times IR \times RAF \times FI \times CF \times EF \times ED}{BW \times AT \times 365 \text{ days/yr}}$$

$$\text{INTAKE-DERMAL} = \frac{CS \times SA \times SAF \times RAF \times CF \times EF \times ED}{BW \times AT \times 365 \text{ days/yr}}$$

Note:

For noncarcinogenic effects: AT =

EF

365 days



TABLE O-76, continued

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (2 - 12 feet)

CONSTRUCTION WORKER

OLEUM PLANT AND POND

BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RPF	INTAKE INGESTION (mg/kg-day)	DERMAL RPF	INTAKE DERMAL (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK DERMAL	TOTAL CANCER RISK
CR	30.3	1	1.4E-07	No values available for Quantitative Analysis		ND			
FB	18	1	9.7E-08			ND			
SUMMARY CANCER RISK							0E+00	0E+00	0E+00

TABLE O-76, continued

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (2 - 12 feet)  
CONSTRUCTION WORKER  
OJITIM PLANT AND POND  
BALJER ARMY AMMUNITION PLANT

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04 - Dec-92

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RPF	INTAKE INGESTION (mg/kg-day)	DERMAL RPF	INTAKE DERMAL (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT DERMAL	TOTAL HAZARD QUOTIENT
CR	30.3	1	2.1E-04	No value available		2.0E-02	0.0104		0.0104
BO	0.115	1	7.9E-07			3.0E-04	0.0026		0.0026
NI	23.1	1	1.6E-04	for Quantitative Analysis		2.0E-02	0.0079		0.0079
NIT	6.19	1	4.2E-05			1.0E-01	0.0004		0.0004
PB	18	1	1.2E-04			ND			
SO4	14000	1	9.6E-02			ND			
SUMMARY HAZARD INDEX							0.0214	0.0000	0.0214

TABLE O-77

INITIALATION EXPOSURE TO AMBIENT AIR  
CONSTRUCTION WORKER  
OILRUM PLANT AND POND  
BADGER ARMY AMMUNITION PLANT

CITY ARW 08-Dec-92

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Median	mg/kg	● zero - 12 feet
CONCENTRATION AIR PARTICULATES	CAp	Calculated	mg/m <sup>3</sup>	see below
CONCENTRATION AIR VOLATILES	CAv	Calculated	mg/m <sup>3</sup>	see below
VOLATILIZATION FACTOR	VF	Calculated	m <sup>3</sup> /kg	Appendix M
24 HOUR AVERAGE PM10 STANDARD	PM10	150	ug/m <sup>3</sup>	USEPA, 1991b
CONVERSION FACTOR	CF	1E-09	kg/ug	
INITIALATION RATE	IR	2.5	m <sup>3</sup> /hour	USEPA, 1991a
BODY WEIGHT	BW	70	kg	USEPA, 1999
EXPOSURE TIME	ET	8	hour/day	Assumption
EXPOSURE FREQUENCY	EF	20	days/year	Assumption
EXPOSURE DURATION	ED	1	years	Assumption
AVERAGING TIME	AT	70	years	USEPA, 1991a
CANCER	AT	0.054794583	years	USEPA, 1991b
NON-CANCER	AT		years	

USEPA, 1999. Risk Assessment Guidelines for Superfund, Part A  
USEPA, 1991a. Standard Default Exposure Factors  
USEPA, 1991b. CFR 50493-07

$$\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$$

$$\text{HAZARD QUOTIENT} = \text{CAp OR CAv (mg/m}^3\text{)} / \text{REFERENCE CONCENTRATION (mg/m}^3\text{)}$$

$$\text{INTAKE} = \frac{(\text{CAp} + \text{CAv}) \times \text{IR} \times \text{ET} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

$$\text{AIR CONCENTRATION PARTICULATES} = \text{CS} \times \text{PM10} \times \text{CF}$$

$$\text{AIR CONCENTRATION VOLATILES} = \text{CS} \times \text{VVF}$$

Note:

For noncarcinogenic effects: AT = EF

365 days

TABLE O-77, continued  
 INHALATION EXPOSURE TO AMBIENT AIR  
 CONSTRUCTION WORKER  
 OILFUM PLANT AND POND  
 BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VF (%/hr)	AIR CONCENTRATION VOLATILES (mg/m <sup>3</sup> )	AIR CONCENTRATION PARTICULATES (mg/m <sup>3</sup> )	ETAEB (mg/kg-day)	CANCER SLOWS FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK
CR	30.3			0.000004545	1.0E-09	4.1E+01	4.2E-06
FB	18			0.0000027	6.0E-10	ND	
SUMMARY CANCER RISK							4E-06

TABLE O-77, continued

INITIALATION EXPOSURE TO AMBIENT AIR  
CONSTRUCTION WORKER  
OLEUM PLANT AND POND  
BADGER ARMY AMMUNITION PLANT

OPPARW

## NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VP (m <sup>3</sup> /kg)	AIR CONCENTRATION VOLATILES (mg/m <sup>3</sup> )	AIR CONCENTRATION PARTICULATES (mg/m <sup>3</sup> )	REFERENCE CONCENTRATION (mg/m <sup>3</sup> )	HAZARD QUOTIENT VOLATILES	HAZARD QUOTIENT PARTICULATES	HAZARD QUOTIENT TOTAL
CR	30.3			0.000004545	ND			
EC	0.115			0.0000000173	8.6E-03		2.0E-04	2E-04
NI	23.1			0.000003455	ND			
MTT	6.19			0.0000009285	ND			
FB	18			0.0000077	ND			
SO4	1400			0.0021	ND			
SUMMARY HAZARD INDEX						0.0000	0.0002	0.0002

TABLE O-78

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SEDIMENT  
 OLDER CHILD (6-16 Years) EXPLORING  
 OLEUM PLANT AND POND  
 BADGER ARMY AMMUNITION PLANT

OPPSST 08 - Dec - 92

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Maximum	mg/kg	USEPA, 1991
INGESTION RATE	IR	100	mg/day	Assumption
FRACTION INGESTED	FI	100%		USEPA, 1992
SOIL ADHERENCE FACTOR	SAF	1	mg/cm <sup>2</sup>	USEPA, 1990
SURFACE AREA EXPOSED	SA	6,150	cm <sup>2</sup> /day	USEPA, 1990
CONVERSION FACTOR	CF	0.000001	kg/mg	Assumption
BODY WEIGHT	BW	40	kg	USEPA, 1990
EXPOSURE FREQUENCY	EF	50	days/year	Assumption
EXPOSURE DURATION	ED	11	years	USEPA, 1989
AVERAGING TIME	AT	70	years	USEPA, 1989
RELATIVE ABSORPTION FACTOR	RAF	11	years	USEPA, 1989
CANCER NONCANCER INGESTION DERMAL		1	unitless	USEPA, 1989
Note: For noncardiac effects: AT = ED For cardiac effects: AT = ED				

USEPA, 1990. Risk Assessment Guidelines for Superfund

USEPA, 1990. Exposure Factors Handbook

USEPA, 1991. Standard Default Exposure Factors

USEPA, 1992. Dermal Exposure Guidelines

TABLE O-78, continued

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SEDIMENT  
 OLDER CHILD (6-16 Years) EXPLORING  
 OLEUM PLANT AND POND  
 BADGER ARMY AMMUNITION PLANT

OPFSST 08-Dec-92

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAV	INTAKE INGESTION (mg/kg-day)	DERMAL RAV	INTAKE DERMAL (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK DERMAL	TOTAL CANCER RISK
No carcinogenic compounds									
SUMMARY CANCER RISK							0E+00	0E+00	0E+00

TABLE O-78, continued

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SEDIMENT  
 OLDER CHILD (6-16 Years) EXPLORING  
 OLEUM PLANT AND POND  
 BADGER ARMY AMMUNITION PLANT

OPPSST 06-Dec-92

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAF	INTAKE INGESTION (mg/kg-day)	DERMAL RAF	INTAKE DERMAL (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT DERMAL	TOTAL HAZARD QUOTIENT
904	500	1	2.0E-04	No values available for Quantitative Analysis		ND	0.0002		0.0002
NET	50	1	1.7E-05			1.0E-01			
SUMMARY HAZARD INDEX							0.0002	0.0000	0.0002



Table O-79  
Compounds Detected  
Ballistics Pond Sediment  
Units: ug/g

Remedial Investigation  
Badger Army Ammunition Plant

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
AL	6 : 6	58000	10200	Y		58000
B2EHP	2 : 6	6.1	1.27	Y		6.1
NH3	5 : 6	215	13.9	Y		215
NIT	1 : 6	5.16	-	Y		5.16
PB	6 : 6	54	2.07	Y		54
PHANTR	1 : 6	0.428	-	Y		0.428
SO4	6 : 6	490	62.7	Y		490

Footnotes:

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential for human nutrition.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum

Note: Assessment of sediment contamination was performed using samples  
BPS-91-01 through BPS-91-06.

**Table O-80**  
**Compounds Detected**  
**Ballistics Pond Surface Water**  
**Units: ug/L**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
AL	2 : 2	180	123	Y		180
BA	2 : 2	36.7	34.6	Y		36.7
CA	2 : 2	6510	6260	N	3	
CL	5 : 5	4050	2934.44	Y		4050
FE	2 : 2	315	217	N	3	
K	2 : 2	1940	1490	N	3	
MG	2 : 2	2920	2810	N	3	
MN	2 : 2	79.1	76.8	Y		79.1
NA	2 : 2	3780	3580	N	3	
NIT	3 : 5	51.4	11.223	Y		51.4
SO4	5 : 5	15000	8516.35	Y		15000
V	1 : 2	5.23	-	Y		5.23
ZN	2 : 2	67.9	35.4	Y		67.9

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant.  
                   \* 3 = essential for human nutrition.  
                   \* 4 = frequency of detection less than 5 %.  
                   \*\* 95th percentile or maximum

Note:           Assessment of surface water contamination was performed using  
                   samples BPW-91-01 and BPW-91-02.

TABLE O-81

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SEDIMENT  
 OLDER CHILD (6-16 Years) EXPLORING  
 BALLISTICS POND  
 BADGER ARMY AMMUNITION PLANT

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Maximum	mg/kg	USEPA, 1991
INGESTION RATE	IR	100	mg/day	Assumption
FRACTION INGESTED	FI	100%		USEPA, 1992
SOIL ADHERENCE FACTOR	SAF	1	mg/cm <sup>2</sup>	USEPA, 1990
SURFACE AREA EXPOSED	SA	6,150	cm <sup>2</sup> /day	Assumption
CONVERSION FACTOR	CF	0.000001	kg/mg	USEPA, 1990
BODY WEIGHT	BW	40	kg	Assumption
EXPOSURE FREQUENCY	EF	50	days/year	Assumption
EXPOSURE DURATION	ED	11	years	Assumption
AVERAGING TIME	AT	70	years	USEPA, 1989
RELATIVE ABSORPTION FACTOR	RAF	11	years	USEPA, 1989
INGESTION		1	unitless	USEPA, 1989
DERMAL		see text		

<p>CANCER RISK = <math>\text{INTAKE} (\text{mg/kg-day}) \times \text{CANCER SLOPE FACTOR} (\text{mg/kg-day})^{-1}</math></p> <p>HAZARD QUOTIENT = <math>\text{INTAKE} (\text{mg/kg-day}) / \text{REFERENCE DOSE} (\text{mg/kg-day})</math></p> <p><math>\text{INTAKE} = (\text{INTAKE-INGESTION}) + (\text{INTAKE-DERMAL})</math></p> <p><math>\text{INTAKE-INGESTION} = \frac{\text{CS} \times \text{IR} \times \text{RAF} \times \text{FI} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ day/yr}}</math></p> <p><math>\text{INTAKE-DERMAL} = \frac{\text{CS} \times \text{SA} \times \text{SAF} \times \text{RAF} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ day/yr}}</math></p>	<p>Note:</p> <p>For noncarcinogenic effects: AT = ED</p>
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USEPA, 1989. Risk Assessment Guidance for Superfund

USEPA, 1990. Exposure Factors Handbook

USEPA, 1991. Standard Default Exposure Factors

USEPA, 1992. Dermal Exposure Guidance

TABLE O-81, continued  
 DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SEDIMENT  
 OLDER CHILD (6-16 Years) EXPLORING  
 BALLISTICS POND  
 BADGER ARMY AMMUNITION PLANT

[HPST]

25-Mar-93

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAP	INTAKE INGESTION (mg/kg-day)	DERMAL RAP	INTAKE DERMAL (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK DERMAL	TOTAL CANCER RISK
BUTYR PB	6.1 54	1 1	3.3E-07 2.9E-06	No values available for Quantitative Analysis		1.4E-02 ND	4.6E-09		4.6E-09
SUMMARY CANCER RISK							5E-09	0E+00	5E-09

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SEDIMENT  
OLDER CHILD (6-16 Years) EXPLORING  
BALLISTICS POND  
RADGER ARMY AMMUNITION PLANT

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAP	INTAKE INGESTION (mg/kg-day)	DERMAL RAP	INTAKE DERMAL (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT DERMAL	TOTAL HAZARD QUOTIENT
AL	5800	1	2.0E-02	No values available for Quantitative Analysis		ND			
BZP	6.1	1	2.1E-06			2.0E-02	1.04E-04		1.04E-04
NH3	215	1	7.4E-05			3.4 mg/l			
NTT	5.16	1	1.8E-06			1.0E-01	1.77E-05		1.77E-05
PB	54	1	1.8E-05			ND			
PICANT R	0.428	1	1.5E-07			4.0E-02	3.66E-05		3.66E-05
SO4	490	1	1.7E-04			ND			
SUMMARY HAZARD INDEX							0.0001	0.0000	0.0001

TABLE 0-82

INGESTION OF AND DERMAL CONTACT WITH SURFACE WATER  
 OLDFATHER CHILD (6 - 16) EXPLORING  
 BALLISTICS POND  
 BADGER ARMY AMMUNITION PLANT

RHSWT

08 - Dec - 92

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION WATER	CW	Maximum	mg/liter	USEPA, 1991
INGESTION RATE	IR	0.05	liters/hour	USEPA, 1990
SURFACE AREA EXPOSED	SA	13,000	cm <sup>2</sup>	USEPA, 1990
BODY WEIGHT	BW	40	kg	USEPA, 1990
CONVERSION FACTOR	CF	0.001	liter/cm <sup>3</sup>	
EXPOSURE TIME	ET	2.6	hours/day	USEPA, 1989
EXPOSURE FREQUENCY	EF	7	days/year	USEPA, 1989
EXPOSURE DURATION	ED	11	years	Assumption
AVERAGING TIME	AT	70	years	USEPA, 1991
CANCER	AT	11	years	USEPA, 1991
NONCANCER	RAF	1	unitless	USEPA, 1989
RELATIVE ABSORPTION FACTOR	PC	0.00155	cm <sup>2</sup> /hour	USEPA, 1992
PERMEABILITY CONSTANT				
USEPA, 1990. Exposure Factors Handbook				
USEPA, 1991. Standard Default Exposure Factors				
USEPA, 1992 Dermal Exposure Assessment				

$$\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$$

$$\text{HAZARD QUOTIENT} = \text{INTAKE (mg/kg-day)} / \text{REFERENCE DOSE (mg/kg-day)}$$

$$\text{INTAKE} = (\text{INTAKE-INGESTION}) + (\text{INTAKE-DERMAL})$$

$$\text{INTAKE-INGESTION} = \frac{\text{CW} \times \text{IR} \times \text{SA} \times \text{CF} \times \text{ET} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

$$\text{INTAKE-DERMAL} = \frac{\text{CW} \times \text{SA} \times \text{PC} \times \text{CF} \times \text{ET} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

Note:

For noncarcinogenic effects: AT = ED

TABLE: O-82, continued

INGESTION OF AND DERMAL CONTACT WITH SURFACE WATER  
 OLDFR CHILD (6 - 16) EXPLORING  
 BALLISTICS POND  
 BADGER ARMY AMMUNITION PLANT

HPSWT 08 - Dec - 92

CARCINOGENIC EFFECTS

COMPOUND	WATER CONCENTRATION (mg/l)	INGESTION EAF	INTAKE INGESTION (mg/kg-day)	PERMEABILITY CONSTANT (cm/hr)	INTAKE DERMAL (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK DERMAL	TOTAL CANCER RISK
No carcinogenic compounds were detected									
SUMMARY CANCER RISK									0E+00

TABLE O-82, continued

INGESTION OF AND DERMAL CONTACT WITH SURFACE WATER  
 OLDHIEP CHILD (6 - 16) EXPLORING  
 BALLISTICS POND  
 RADGER ARMY AMMUNITION PLANT

BFSWT

08 - Dec - 92

NONCARCINOGENIC EFFECTS

COMPOUND	WATER CONCENTRATION (mg/l)	INGESTION RPF	INGESTION (mg/kg-day)	PERMEABILITY CONSTANT (K) (cm/hr)	INTAKE DERMAL (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT DERMAL	TOTAL HAZARD QUOTIENT
AL	0.18	1	4.3E-06	0.00155	4.7E-06	ND	1.3E-05	1.3E-05	2.6E-05
BA	0.0307	1	8.8E-07	0.00155	9.2E-07	7.0E-02	1.3E-05	1.3E-05	
CL	4.05	1	9.7E-05	0.00155	1.0E-04	ND	1.9E-05	2.0E-05	3.9E-05
MM	0.0791	1	1.9E-06	0.00155	2.0E-06	1.0E-01	1.2E-05	1.3E-05	2.5E-05
NTT	0.0514	1	1.2E-06	0.00155	1.3E-06	1.0E-01	1.2E-05	1.3E-05	
904	15	1	3.6E-04	0.00155	3.8E-04	ND	1.8E-05	1.9E-05	3.7E-05
V	0.00523	1	1.3E-07	0.00155	1.3E-07	7.0E-03	8.1E-06	8.5E-06	1.7E-05
ZN	0.0679	1	1.6E-06	0.00155	1.7E-06	2.0E-01			
SUMMARY HAZARD INDEX									0.00014



**Table O-83**  
**Compounds Detected**  
**Old Acid Area Surface Soil (0-2')**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
CR	3 : 3	20.5	14.4	N	1	
MEK	2 : 3	0.006	-	N	2	
NI	3 : 3	56.9	17.3	Y		56.9
NIT	13 : 23	5.61	1.09	Y		1.79
PB	3 : 3	1500	4.87	Y		1500
SO4	16 : 23	20000	5.78	Y		18000

Footnotes: \* 1 = within background range.  
\* 2 = laboratory or sampling contaminant.  
\* 3 = essential for human nutrition.  
\* 4 = frequency of detection less than 5 %.  
\*\* 95th percentile or maximum

Note: Assessment of surface soil contamination (0 to 2 feet) was performed using samples from borings OAB-91-01 through OAB-91-13.

Table O-84  
Compounds Detected  
Old Acid Area Subsurface Soil (0-12")  
Units: ug/g

**Remedial Investigation  
Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
ACET	2 : 6	0.008	0.004	Y		0.008
CH2CL2	1 : 6	0.002	-	N	2	
CR	9 : 9	20.5	2.4	Y		20.5
MEK	6 : 9	0.008	0.006	N	2	
NI	9 : 9	17.3	4.81	Y		17.3
NIT	19 : 29	8.28	1.09	Y		8.28
PB	6 : 6	1500	3.03	Y		1500
SO4	22 : 29	20000	5.78	Y		20000

Footnotes:

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential for human nutrition.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum

Note: Assessment of subsurface soil contamination (0 to 12 feet) was performed using samples from borings OAB-91-01 through OAB-91-13.

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
OLD ACID AREA  
BADGER ARMY AMMUNITION PLANT

CLASSIFICATION 25 - Mar - 93

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Medium	mg/kg	USEPA, 1991
INGESTION RATE - ADULT	IRa	100	mg/day	USEPA, 1991
INGESTION RATE - CHILD	IRc	200	mg/day	USEPA, 1991
FRACTION INGESTED	FI	100%		Assumption
CONVERSION FACTOR	CF	0.000001	kg/mg	
BODY WEIGHT - ADULT	BWa	70	kg	USEPA, 1991
BODY WEIGHT - CHILD	BWc	15	kg	USEPA, 1991
EXPOSURE FREQUENCY	EF	350	days/year	USEPA, 1991
EXPOSURE DURATION - ADULT	EDA	24	years	USEPA, 1991
EXPOSURE DURATION - CHILD	EDc	6	years	USEPA, 1991
AVERAGING TIME				
CANCER	AT	70	years	USEPA, 1989
ADULT - NONCANCER	ATa	24	years	USEPA, 1991
CHILD - NONCANCER	ATc	6	years	USEPA, 1991
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	USEPA, 1989

USEPA, 1989. Risk Assessment Guidelines for Superfund  
USEPA, 1991. Standard Default Exposure Factors

Note:

For cancer diagnostic effects: AT = ED

$$\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$$

$$\text{HAZARD QUOTIENT} = \text{INTAKE (mg/kg-day)} / \text{REFERENCE DOSE (mg/kg-day)}$$

$$\text{INTAKE-ADULT} = \frac{\text{CS} \times \text{IRa} \times \text{FI} \times \text{CF} \times \text{PE} \times \text{EDa}}{\text{BWa} \times \text{ATa} \times \text{SAS day/yr}}$$

$$\text{INTAKE-CHILD} = \frac{\text{CS} \times \text{IRc} \times \text{FI} \times \text{CF} \times \text{PE} \times \text{EDc}}{\text{BWc} \times \text{ATc} \times \text{SAS day/yr}}$$

TABLE O-85, continued  
INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
OLD ACID AREA  
BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION R/F	INTAKE ADULT (mg/kg-day)	INTAKE CHILD (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK ADULT	CANCER RISK CHILD	TOTAL CANCER RISK
PB	1500	1	7.0E-04	1.4E-03	ND			
SUMMARY CANCER RISK						9E+00	9E+00	9E+00

TABLE O-85, continued

INCIDENTAL INGESTION OF SURFACE SOIL  
RESIDENTIAL - ADULT AND CHILD  
OLD ACID AREA  
BADGER ARMY AMMUNITION PLANT

## NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION BAF	INTAKE		REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT		TOTAL HAZARD QUOTIENT
			ADULT (mg/kg-day)	CHILD (mg/kg-day)		ADULT	CHILD	
MI	56.9	1	7.8E-05	7.5E-04	2.0E-02	0.0039	0.0364	0.0403
NTT	1.79	1	2.5E-06	2.5E-05	1.0E-01	0.0000	0.0002	0.0003
PS	1500	1	2.1E-03	1.9E-02	ND			
SO4	18000	1	2.5E-02	2.5E-01	ND			
SUMMARY HAZARD INDEX						0.004	0.037	0.041

INCIDENTAL INGESTION AND INHALATION OF SOIL  
GROUNDS MAINTENANCE WORKPIR  
OLD ACID AREA  
RADGER ARMY AMMUNITION PLANT

OASGW

04-Dec-92

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Medium	mg/kg	USEPA, 1991a
INGESTION RATE	IR	100	mg/day	Assumption
FRACTION INGESTED	FI	100%		
CONVERSION FACTOR	CF	0.00001	kg/mg	
BODY WEIGHT	BW	70	kg	USEPA, 1991a
EXPOSURE FREQUENCY	EF	24	days/year	USEPA, 1991a
EXPOSURE DURATION	ED	25	years	USEPA, 1991a
CONCENTRATION AIR PARTICULATES	Cap	Calculated	mg/m <sup>3</sup>	Appendix M
CONCENTRATION AIR VOLATILES	CAV	Calculated	m <sup>3</sup> /kg	USEPA, 1991b
VOLATILIZATION FACTOR	VF	Calculated	m <sup>3</sup> /kg	USEPA, 1991a
PARTICULATE EMISSION FACTOR	PEF	4.63E+09	m <sup>3</sup> /hour	Assumption
INHALATION RATE	IhR	2.5	m <sup>3</sup> /hour	
EXPOSURE TIME	ET	6	hours/day	
AVERAGING TIME	AT	70	years	USEPA, 1989
	AT	25	years	USEPA, 1991a
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	USEPA, 1989
CANCER				
NONCANCER				
RELATIVE ABSORPTION FACTOR				

USEPA, 1989. Risk Assessment Guidelines for Superfund-Part A

USEPA, 1990. Exposure Factors Handbook

USEPA, 1991b. Risk Assessment Guidelines for Superfund-Part B

USEPA, 1991a. Standard Default Exposure Factors

$$\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$$

$$\text{HAZARD QUOTIENT}_{\text{Ingestion}} = \text{INTAKE (mg/kg-day)} / \text{REFERENCE DOSE (mg/kg-day)}$$

$$\text{HAZARD QUOTIENT}_{\text{Inhalation}} = \frac{\text{AIR CONCENTRATION (mg/m}^3\text{)}}{\text{REFERENCE CONCENTRATION (mg/m}^3\text{)}}$$

$$\text{INTAKE-INGESTION} = \frac{\text{CS} \times \text{IR} \times \text{RAF} \times \text{FI} \times \text{CF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

$$\text{INTAKE-INHALATION} = \frac{(\text{Cap} + \text{CAV}) \times \text{IhR} \times \text{ET} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

$$\text{AIR CONCENTRATION (mg/m}^3\text{)} = \text{Cap} + \text{CAV}$$

$$\text{Cap} = \text{CS} \times \text{IhREF}$$

$$\text{CAV} = \text{CS} \times \text{IhVP}$$

Note:

For noncarcinogenic effects: AT = ED

TABLE O-86, continued

INCIDENTAL INGESTION AND INHALATION OF SOIL  
GROUNDS MAINTENANCE WORKER  
OLD ACID AREA  
BADGER ARMY AMMUNITION PLANT

OASS(JW) 08-Dec-92

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RPF	INTAKE INGESTION (mg/kg-day)	INTAKE INHALATION (mg/kg-day)	CANCER SLOPE FACTOR-ING. (mg/kg-day) <sup>-1</sup>	CANCER SLOPE FACTOR-INO. (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK INHALATION	TOTAL CANCER RISK
PB	1500	1	5.0E-05	2.2E-09	ND	ND			
SUMMARY CANCER RISK									0E+00

TABLE O-84, continued

0/ASSOW 08-Dec-92

INCIDENTAL INGESTION AND INHALATION OF SOIL  
 GROUNDS MAINTENANCE WORKER  
 OLD ACID AREA  
 BADGER ARMY AMMUNITION PLANT

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION BAF	INTAKE (mg/kg-day)	AIR CONCENT. (mg/m <sup>3</sup> )	REFERENCE CONCENT. (mg/m <sup>3</sup> )	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT INHALATION	TOTAL HAZARD QUOTIENT
NI	56.9	1	5.3E-06	1.2E-08	ND	2.0E-02	0.0003		0.0003
NIT	1.79	1	1.7E-07	3.9E-10	ND	1.0E-01	0.0000		0.0000
PB	1500	1	1.4E-04	3.2E-07	ND	ND			
SO4	18000	1	1.7E-03	3.9E-06	ND	ND			
SUMMARY HAZARD INDEX									0.0003



TABLE O-87

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0 - 12 feet)  
CONSTRUCTION WORKER  
OLD ACID AREA  
BADGER ARMY AMMUNITION PLANT

OASB-CW

08-1 Dec-92

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Maximum	mg/kg	USEPA, 1991
INGESTION RATE	IR	480	mg/day	Assumption
FRACTION INGESTED	FI	100%		USEPA, 1992
SOIL ADHERENCE FACTOR	SAF	1	mg/cm <sup>2</sup>	USEPA, 1990
SURFACE AREA EXPOSED	SA	2,100	cm <sup>2</sup> /day	USEPA, 1990
CONVERSION FACTOR	CF	0.00001	kg/mg	
BODY WEIGHT	BW	70	kg	USEPA, 1991
EXPOSURE FREQUENCY	EF	20	days/year	Assumption
EXPOSURE DURATION	ED	1	years	Assumption
AVERAGING TIME	AT	70	years	USEPA, 1989
CANCER	AT	0.054794203	days	Assumption
NONCANCER	AT			
RELATIVE ABSORPTION FACTOR	RAF			
INGESTION				
DERMAL		1	unitless	USEPA, 1989

$$\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$$

$$\text{HAZARD QUOTIENT} = \text{INTAKE (mg/kg-day)} / \text{REFERENCE DOSE (mg/kg-day)}$$

$$\text{INTAKE} = (\text{INTAKE-INGESTION}) + (\text{INTAKE-DERMAL})$$

$$\text{INTAKE-INGESTION} = \frac{CS \times IR \times RAF \times FI \times CF \times EF \times ED}{BW \times AT \times 365 \text{ days/yr}}$$

$$\text{INTAKE-DERMAL} = \frac{CS \times SA \times SAF \times RAF \times CF \times EF \times ED}{BW \times AT \times 365 \text{ days/yr}}$$

Note:

For noncarcinogenic effects: AT =

EF

365 days

USEPA, 1989, Risk Assessment Guidance for Superfund

USEPA, 1990, Exposure Factors Handbook

USEPA, 1991, Standard Default Exposure Factors

USEPA, 1992, Dermal Absorption Guidelines

TABLE 0-87, continued  
DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0 - 12 feet)  
CONSTRUCTION WORKER  
OLD ACID AREA  
PADGER ARMY AMMUNITION PLANT

OASH-CW 08-DNC-92

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION BAF	INTAKE INGESTION (mg/kg-day)	DERMAL BAF	INTAKE DERMAL (mg/kg-day)	CANCER FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK DERMAL	TOTAL CANCER RISK
CR	20.5	1	1.1E-07	No values available for Quantitative Analysis		ND			
PS	1500	1	8.1E-06			ND			
SUMMARY CANCER RISK							0E+00	0E+00	0E+00

TABLE U-87, continued

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (0 - 12 feet)  
CONSTRUCTION WORKER  
OLD ACID AREA  
BADGER ARMY AMMUNITION PLANT

OASB-CW

08-Dec-92

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAP	INTAKE (mg/kg-day)	DERMAL RAP	DERMAL DPERMAL (mg/kg-day)	REFERENCE Dose (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT DERMAL	TOTAL HAZARD QUOTIENT
ALBT	0.008	1	5.5E-06	No values available		1.0E+00	5.49E-06		5.49E-06
CR	20.5	1	1.4E-04	available for		2.0E-02	7.03E-06		7.03E-06
PI	17.3	1	1.2E-04	Quantitative Analysis		2.0E-02	5.93E-06		5.93E-06
NTT	8.28	1	5.7E-05			1.0E-01	5.68E-04		5.68E-04
FB	1500	1	1.0E-02			ND			
904	20000	1	1.4E-01			ND			
SUMMARY HAZARD INDEX							0.0135	0.0000	0.0135

INITIATION EXPOSURE TO AMBIENT AIR  
CONSTRUCTION WORKER  
OLD ACID AREA  
BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS

EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL				0 zero - 12 feet see below
CONCENTRATION AIR PARTICULATES	CS	Medium Calculated	mg/kg	see below
CONCENTRATION AIR VOLATILES	CAV	Calculated	mg/m <sup>3</sup>	see below
VOLATILIZATION FACTOR	VF	Calculated	m <sup>3</sup> /kg	Appendix M
24 HOUR AVERAGE PM10 STANDARD	PM10	150	ug/m <sup>3</sup>	USEPA 1991b
CONVERSION FACTOR	CF	1E-09	kg/ug	
INITIATION RATE	IR	2.5	m <sup>3</sup> /hour	USEPA 1991a
BODY WEIGHT	BW	70	kg	USEPA 1989
EXPOSURE TIME	ET	8	hour/day	Assumption
EXPOSURE FREQUENCY	EF	20	days/year	Assumption
EXPOSURE DURATION	ED	1	years	Assumption
AVERAGING TIME				
CANCER	AT	70	years	USEPA 1991a
NONCANCER	AT	0.0547945705	years	USEPA 1991a
USEPA 1989 Risk Assessment Guidelines for Superfund Part A				
USEPA 1991a Standard Default Exposure Factors				
USEPA 1991b CFR 50493-097				

$$\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$$

$$\text{HAZARD QUOTIENT} = \text{CAV OR CAV (mg/m}^3\text{)} / \text{REFERENCE CONCENTRATION (mg/m}^3\text{)}$$

$$\text{INTAKE} = \frac{(\text{CAV} \times \text{CAV}) \times \text{IR} \times \text{ET} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

$$\text{AIR CONCENTRATION PARTICULATES} = \text{CS} \pm \text{PM10} \pm \text{CF}$$

$$\text{AIR CONCENTRATION VOLATILES} = \text{CS} \pm \text{IVF}$$

Note:

For noncardiopulmonary effects: AT = EF 365 days

TABLE O-14, continued  
INITIALATION EXPOSURE TO AMBIENT AIR  
CONSTRUCTION WORKER  
OLD ACID AREA  
BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VF (m <sup>3</sup> /kg)	AIR CONCENTRATION VOLATILES (mg/m <sup>3</sup> )	AIR CONCENTRATION PARTICULATES (mg/m <sup>3</sup> )	INTAKE (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK
CR	17.5			0.0000264	5.9E-10	4.1E+01	2.4E-08
PS	240			0.000036	8.1E-09		
SUMMARY CANCER RISK							2E-08

TAB J: O-8A, continued  
 INHALATION EXPOSURE TO AMBIENT AIR  
 CONSTRUCTION WORKER  
 OLD ACID AREA  
 BADGER ARMY AMMUNITION PLANT

UAAW

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VF (m³/kg)	AIR CONCENTRATION VOLATILES (mg/m³)	AIR CONCENTRATION PARTICULATES (mg/m³)	REFERENCE CONCENTRATION (mg/kg a)	HAZARD QUOTIENT VOLATILES	HAZARD QUOTIENT PARTICULATES	HAZARD QUOTIENT TOTAL
ACET	0.008			0.0000000012	ND			
CE	17.6			0.00000264	ND			
ME	17.3			0.000002595	ND			
MTT	8.28			0.000001241	ND			
PS	240			0.0000036	ND			
SO4	2500			0.0000375	ND			
SUMMARY HAZARD INDEX								0

Table O-89  
Compounds Detected  
Old Fuel Oil Tank Area Subsurface Soil (2'-12')  
Units: ug/g

Remedial Investigation  
Badger Army Ammunition Plant

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	<u>Concentration **</u>
2MNAP	1: 6	1.07	-	Y		1.07
ANAPNE	1: 6	0.077	-	Y		0.077
B2EHP	2: 6	1.8	1.23	Y		1.8
BAANTR	3: 6	0.122	0.08	Y		0.122
BGHIPY	1: 6	0.396	-	Y		0.396
CHRY	2: 6	0.113	0.076	Y		0.113
DNBP	1: 6	2.1	-	Y		2.1
FANT	1: 6	0.037	-	Y		0.037
FLRENE	1: 6	0.16	-	Y		0.16
PHANTR	2: 6	0.194	0.088	Y		0.194

Footnotes:

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential for human nutrition.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum

Note: Assessment of subsurface soil contamination (2 to 12 feet)  
was performed using samples from FTB-91-01 and FTB-91-02.

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (2 - 12 feet)  
CONSTRUCTION WORKER  
OLD FUEL OIL TANK  
BADGER ARMY AMMUNITION PLANT

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Medium	mg/kg	USEPA, 1991
INGESTION RATE	IR	480	mg/day	Assumption
FRACTION INGESTED	FI	100%		USEPA, 1992
SOIL ADHERENCE FACTOR	SAF	1	mg/cm <sup>2</sup>	USEPA, 1990
SURFACE AREA EXPOSED	SA	2,100	cm <sup>2</sup> /day	USEPA, 1990
CONVERSION FACTOR	CF	0.000001	kg/mg	Assumption
BODY WEIGHT	BW	70	kg	USEPA, 1991
EXPOSURE FREQUENCY	EF	20	days/year	Assumption
EXPOSURE DURATION	ED	1	years	Assumption
AVERAGING TIME	AT	70	years	USEPA, 1989
CANCER	AT	0.054794205	years	Assumption
NONCANCER	AT		years	Assumption
RELATIVE ABSORPTION FACTOR	RAF		unitless	USEPA, 1989
INGESTION				
DERMAL				

USEPA, 1989. Risk Assessment Guidelines for Superfund

USEPA, 1990. Exposure Factors Handbook

USEPA, 1991. Standard Default Exposure Factors

USEPA, 1992. Dermal Absorption Guidelines

CANCER RISK = INTAKE (mg/kg-day) x CANCER SLOPE FACTOR (mg/kg-day)<sup>-1</sup>

HAZARD QUOTIENT = INTAKE (mg/kg-day) / REFERENCE DOSE (mg/kg-day)

INTAKE = (INTAKE-INGESTION) + (INTAKE-DERMAL)

INTAKE-INGESTION =  $\frac{CS \times IR \times RAF \times FI \times CF \times EF \times ED}{BW \times AT \times 365 \text{ days/yr}}$ INTAKE-DERMAL =  $\frac{CS \times SA \times SAF \times RAF \times CF \times EF \times ED}{BW \times AT \times 365 \text{ days/yr}}$ 

Note:

For cancer diagnostic effects: AT =

EF

365 days



TABLE O-90, continued

DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (2 - 12 feet)  
CONSTRUCTION WORKER  
OLD FUEL OIL TANK  
BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAF	INTAKE INGESTION (mg/kg-day)	DERMAL RAF	INTAKE DERMAL (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK DERMAL	TOTAL CANCER RISK
BZESBP	1.8	1	9.7E-09	No values available for Quantitative Analysis		1.4E-02	1.4E-10		1.4E-10
BAAHTR	0.122	1	6.3E-10			7.3E+00	4.8E-09		4.8E-09
CHRY	0.113	1	6.1E-10			7.3E+00	4.4E-09		4.4E-09
SUMMARY CANCER RISK							9E-09	0E+00	9E-09

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Rev. 8/92

TABLE O-90, continued  
DERMAL CONTACT WITH AND INCIDENTAL INGESTION OF SOIL (2 - 12 feet)  
CONSTRUCTION WORKER  
OLD FUEL OIL TANK  
BADGER ARMY AMMUNITION PLANT

DOTSD-CW 08-Dec-92

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	INGESTION RAI	INTAKE INGESTION (mg/kg-day)	DERMAL RAI	INTAKE DERMAL (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT DERMAL	TOTAL HAZARD QUOTIENT
ZINC	1.07	1	7.3E-06	No values available for Quantitative Analysis		4.0E-02	1.83E-04		1.83E-04
ANALINE	0.077	1	5.3E-07			4.0E-02	1.32E-05		1.32E-05
BIPHE	1.8	1	1.2E-05			2.0E-02	6.17E-04		6.17E-04
BAXTR	0.122	1	8.4E-07			4.0E-02	2.09E-05		2.09E-05
CHRY	0.396	1	2.7E-06			4.0E-02	6.79E-05		6.79E-05
CHRY	0.113	1	7.7E-07			4.0E-02	1.94E-05		1.94E-05
DNBP	2.1	1	1.4E-05			1.0E+00	1.44E-05		1.44E-05
PANT	0.037	1	2.5E-07			4.0E-01	6.34E-07		6.34E-07
PLENE	0.16	1	1.1E-06			4.0E-01	2.74E-06		2.74E-06
PLANTR	0.194	1	1.3E-06			4.0E-02	3.33E-05		3.33E-05
SUMMARY HAZARD INDEX							0.0010	0.0000	0.0010

TABLE O-91

INITIATION EXPOSURE TO AMBIENT AIR  
CONSTRUCTION WORKER  
OLD FUEL OIL TANK  
BADGER ARMY AMMUNITION PLANT

FOIA b 7  
08-Dec-92

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION SOIL	CS	Maximum Calculated	mg/kg	@ 100 - 12 feet see below
CONCENTRATION AIR PARTICULATES	CAp	Calculated	mg/m <sup>3</sup>	see below
CONCENTRATION AIR VOLATILES	CAV	Calculated	mg/m <sup>3</sup>	Appendix M
VOLATILIZATION FACTOR	VF	Calculated	m <sup>3</sup> /kg	USEPA, 1991b
24 HOUR AVERAGE PM10 STANDARD	PM10	150	ug/m <sup>3</sup>	
CONVERSION FACTOR	CF	1E-09	kg/ug	
INHALATION RATE	InR	2.5	m <sup>3</sup> /hour	USEPA, 1991a
BODY WEIGHT	BW	70	kg	USEPA, 1989
EXPOSURE TIME	ET	8	hours/day	Assumption
EXPOSURE FREQUENCY	EF	20	days/year	Assumption
EXPOSURE DURATION	ED	1	years	Assumption
AVERAGING TIME	AT	70	years	USEPA, 1991a
CANCER	AT	0.054794503	years	USEPA, 1991a
NONCANCER	AT			

USEPA, 1989, Risk Assessment Guidance for Superfund, Part A  
 USEPA, 1991a, Standard Default Exposure Factors  
 USEPA, 1991b, CFR 505.503 - 507

CANCER RISK = INTAKE (mg/kg-day) x CANCER SLOPE FACTOR (mg/kg-day)<sup>-1</sup> - 1  
 HAZARD QUOTIENT = CAp OR CAV (mg/cm<sup>3</sup>) / REFERENCE CONCENTRATION (mg/cm<sup>3</sup>)  
 INTAKE = (CAp x CAV) x InR x ET x EF x ED  
 BW x AT x 365 days/yr  
 AIR CONCENTRATION PARTICULATES = CS x PM10 x CF  
 AIR CONCENTRATION VOLATILES = CS x 1/VF  
 Note:  
 For noncarcinogenic effects: AT = 365 days

TABLE O-91, continued  
INITIALATION EXPOSURE TO AMBIENT AIR  
CONSTRUCTION WORKER  
OLD FUEL OIL TANK  
BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VP (m³/kg)	AIR CONCENTRATION VOLATILES (mg/m³)	AIR CONCENTRATION PARTICULATES (mg/m³)	INTAKE (mg/kg-day)	CANCER SLOPE FACTOR (mg/kg-day)⁻¹	CANCER RISK
BENZP	1.8			0.00000027	6.0E-11	ND	
BAAHTR	0.122			0.000000083	4.1E-12	6.1E+00	2.5E-11
CHRY	0.113			0.000000017	3.8E-12	6.1E+00	2.3E-11
SUMMARY CANCER RISK							5E-11

TABLE O-91, continued  
 INITIALATION EXPOSURE TO AMBIENT AIR  
 CONSTRUCTION WORKER  
 OLD FUEL OIL TANK  
 BADGER ARMY AMMUNITION PLANT

NONCARCINOGENIC EFFECTS

COMPOUND	SOIL CONCENTRATION (mg/kg)	VF ( $\mu$ g)	AIR CONCENTRATION		REFERENCE CONCENTRATION (mg/m <sup>3</sup> )	HAZARD QUOTIENT		HAZARD QUOTIENT TOTAL
			VOLATILES ( $\mu$ g/m <sup>3</sup> )	PARTICULATES ( $\mu$ g/m <sup>3</sup> )		VOLATILES	PARTICULATES	
2,4-DAP	1.07			0.000001605	ND			
ANAPNE	0.077			0.000000016	ND			
BZEP	1.8			0.00000027	ND			
BAANTR	0.122			0.000000083	ND			
BCREPT	0.396			0.000000094	ND			
CEBY	0.113			0.000000017	ND			
DNEP	2.1			0.000000015	ND			
PANT	0.037			0.000000056	ND			
FLREPE	0.16			0.000000024	ND			
FLANTR	0.194			0.000000091	ND			
SUMMARY HAZARD INDEX								
						0	0	0

INGESTION OF AND INHALATION OF VAPORS FROM HOUSEHOLD WATER<sup>1</sup>

RESIDENTIAL - ADULT

OFP - POST WELLS

BATKIER ARMY AMMUNITION PLANT

## EXPOSURE PARAMETERS

## EQUATIONS

PARAMETER	SYMBOL	VALUE	UNITS	SOURCE
CONCENTRATION WATER	CW		mg/liter	USEPA, 1991a
INGESTION RATE	IR	2	liters/day	USEPA, 1990
INHALATION RATE	IH	15	m <sup>3</sup> /day	USEPA, 1989
BODY WEIGHT	BW	70	kg	USEPA, 1991b
VOLATILIZATION CONSTANT	K	0.5	L/m <sup>3</sup> <sup>2</sup>	USEPA, 1991a
EXPOSURE FREQUENCY	EF	350	days/year	USEPA, 1991a
EXPOSURE DURATION	ED	24	years	USEPA, 1991a
AVERAGING TIME	AT	70	years	USEPA, 1991a
	AT	24	years	USEPA, 1991a
RELATIVE ABSORPTION FACTOR	RAF	1	unitless	Assumption

USEPA, 1989, Risk Assessment Guidelines for Superfund

USEPA, 1991a, Standard Default Exposure Factors

USEPA, 1991b, Risk Assessment Guidelines for Superfund, Volume 1, Part B

## Note:

For noncarcinogenic effects: AT = ED

<sup>1</sup> Household use includes laundering, dishwashing, and showering<sup>2</sup> Applied only to chemicals with a Henry's Law Constant > 1x10<sup>-5</sup> atm-cm<sup>3</sup>/mole and a molecular weight of less than 200 g/mole

$$\text{CANCER RISK} = \text{INTAKE (mg/kg-day)} \times \text{CANCER SLOPE FACTOR (mg/kg-day)}^{-1}$$

$$\text{HAZARD QUOTIENT} = \text{INTAKE (mg/kg-day)} / \text{REFERENCE DOSE (mg/kg-day)}$$

$$\text{INTAKE} = (\text{INTAKE-INGESTION}) + (\text{INTAKE-INHALATION})$$

$$\text{INTAKE-INGESTION} = \frac{\text{CW} \times \text{IR} \times \text{RAF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

$$\text{INTAKE-INHALATION} = \frac{\text{CW} \times \text{K} \times \text{IH} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT} \times 365 \text{ days/yr}}$$

TABLE O-92, continued

INGESTION OF AND INHALATION OF VAPORS FROM HOUSEHOLD WATER!  
RESIDENTIAL - ADULT  
OPP - POST WELLS  
BADGER ARMY AMMUNITION PLANT

CARCINOGENIC EFFECTS

COMPOUND	WATER CONCENTRATION (mg/l)	INTAKE INGESTION (mg/kg-day)	INTAKE INHALATION (mg/kg-day)	ORAL SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	INHALATION SLOPE FACTOR (mg/kg-day) <sup>-1</sup>	CANCER RISK INGESTION	CANCER RISK INHALATION	TOTAL CANCER RISK
CYLA	0.0108	1.0E-04	3.8E-04	0.13	5.2E-02	1.3E-03	2.0E-03	3.3E-03
CMTJ	0.00131	1.2E-03	4.6E-03	0.0061	8.1E-03	7.3E-08	3.7E-07	4.3E-07
TRCLR	0.000425	4.0E-06	1.3E-03	0.011	1.7E-02	4.4E-08	2.5E-07	3.0E-07
SUMMARY CANCER RISK								
						1E-05	2E-05	3E-05

TABLE O-92, continued  
 INGESTION OF AND INHALATION OF VAPORS FROM HOUSEHOLD WATER!  
 RESIDENTIAL - ADULT  
 OFF-POST WELLS  
 BADGER ARMY AMMUNITION PLANT

## NONCARCINOGENIC EFFECTS - Version 1

COMPOUND	WATER CONCENTRATION (mg/l)	INTAKE INGESTION (mg/kg-day)	INTAKE INHALATION (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT INHALATION	TOTAL HAZARD QUOTIENT
CCL4	0.0108	1.0E-04	3.8E-04	0.0007	1.4E-01	Reference Dose Not Available for Inhalation Route	1.4E-01
CHCL3	0.00131	1.2E-03	4.6E-03	0.01	1.2E-03		1.2E-03
TRICL3	0.000423	4.0E-06	1.5E-05	ND			
BA	0.0248	2.3E-04	8.7E-04	0.07	3.3E-03		3.3E-03
CR	0.0145	1.4E-04	5.1E-04	0.003	2.7E-02		2.7E-02
MON	0.0341	5.1E-04	1.9E-03	0.1	5.1E-03		5.1E-03
MTT, as NO2	27	2.5E-01	9.5E-01	0.1	2.5E+00		2.5E+00
CD	0.00278	2.6E-05	9.8E-05	0.0005	5.2E-02		5.2E-02
SUMMARY HAZARD INDEX					3	0	3

## NONCARCINOGENIC EFFECTS - Version 2

COMPOUND	WATER CONCENTRATION (mg/l)	INTAKE INGESTION (mg/kg-day)	INTAKE INHALATION (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT INGESTION	HAZARD QUOTIENT INHALATION	TOTAL HAZARD QUOTIENT
CCL4	0.0108	1.0E-04	3.8E-04	0.0007	1.4E-01	Reference Dose Not Available for Inhalation Route	1.4E-01
CHCL3	0.00131	1.2E-03	4.6E-03	0.01	1.2E-03		1.2E-03
TRICL3	0.000423	4.0E-06	1.5E-05	ND			
BA	0.0248	2.3E-04	8.7E-04	0.07	3.3E-03		3.3E-03
CR	0.0145	1.4E-04	5.1E-04	0.003	2.7E-02		2.7E-02
MON	0.0341	5.1E-04	1.9E-03	0.1	5.1E-03		5.1E-03
MTT, as NO2	27	2.5E-01	9.5E-01	1.6	1.6E-01		1.6E-01
CD	0.00278	2.6E-05	9.8E-05	0.0005	5.2E-02		5.2E-02
SUMMARY HAZARD INDEX					0.39	0.00	0.39



## **BIBLIOGRAPHY FOR APPENDIX O**

### **REMEDIAL INVESTIGATION BADGER ARMY AMMUNITION PLANT**

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**APPENDIX P**  
**INVENTORY OF SITE SPECIES**

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**SPECIES PROFILES AND HABITAT REQUIREMENTS  
OF THREATENED AND ENDANGERED SPECIES KNOWN  
TO OCCUR IN THE VICINITY OF BAAP**

The following species profiles have been compiled to provide relevant information concerning the range, habitat preference, and foraging behavior of a number of special status species that are known to occur in the general vicinity of Badger Army Ammunition Plant. It should be stressed that none of the species has been documented as occurring at the facility, however this information has been used to evaluate the potential for exposure.

**I. AVIFAUNA**

**Cooper's Hawk (*Accipiter cooperii*)**

The Cooper's hawk breeds from Nova Scotia to western Canada, south to Florida and the Gulf Coast; the overwintering range extends from southern New England and west, including southern and central Wisconsin, and south to Central America (DeGraaf and Rudis, 1986; Peterson, 1980). This hawk nests in trees located in wooded forests or swamps interspersed with open fields. The Cooper's Hawk forages for small to medium sized birds, mammals, and amphibians in open fields and near forest edges. The typical territory size does not extend farther than 1 mile from the nest (DeGraaf and Rudis, 1986).

The Cooper's Hawk has been observed in the vicinity of Mud Lake, which is located approximately six miles southeast of BAAP and two miles from the Prairie Du Sac section of the Wisconsin river (see figure P-1). This bird has also been observed in the Pine Glen area, which borders the north side of BAAP. The habitats at Mud Lake, Pine Glen, and BAAP all feature open areas with intermittent vegetation. Given this and the close proximity of Pine Glen to BAAP (within one mile), it is likely that Cooper's hawks observed at Pine Glen may also be found at BAAP, particularly in the northern half of the facility.

**Kentucky Warbler (*Oporonis formosus*), Hooded Warbler (*Wilsonia citrina*), Worm Eating Warbler (*Helminthos vermivorus*)**

These birds breed in the southeastern U.S and winter in Mexico and the West Indies. Southern Wisconsin forms the northernmost boundary of their summer nesting (Peterson, 1980). Wooded slopes and wetlands vegetation surrounded by second growth understory are required for nesting. These three insectivorous birds all forage on the forest floor and within thicketed areas (DeGraaf and Rudis, 1986).

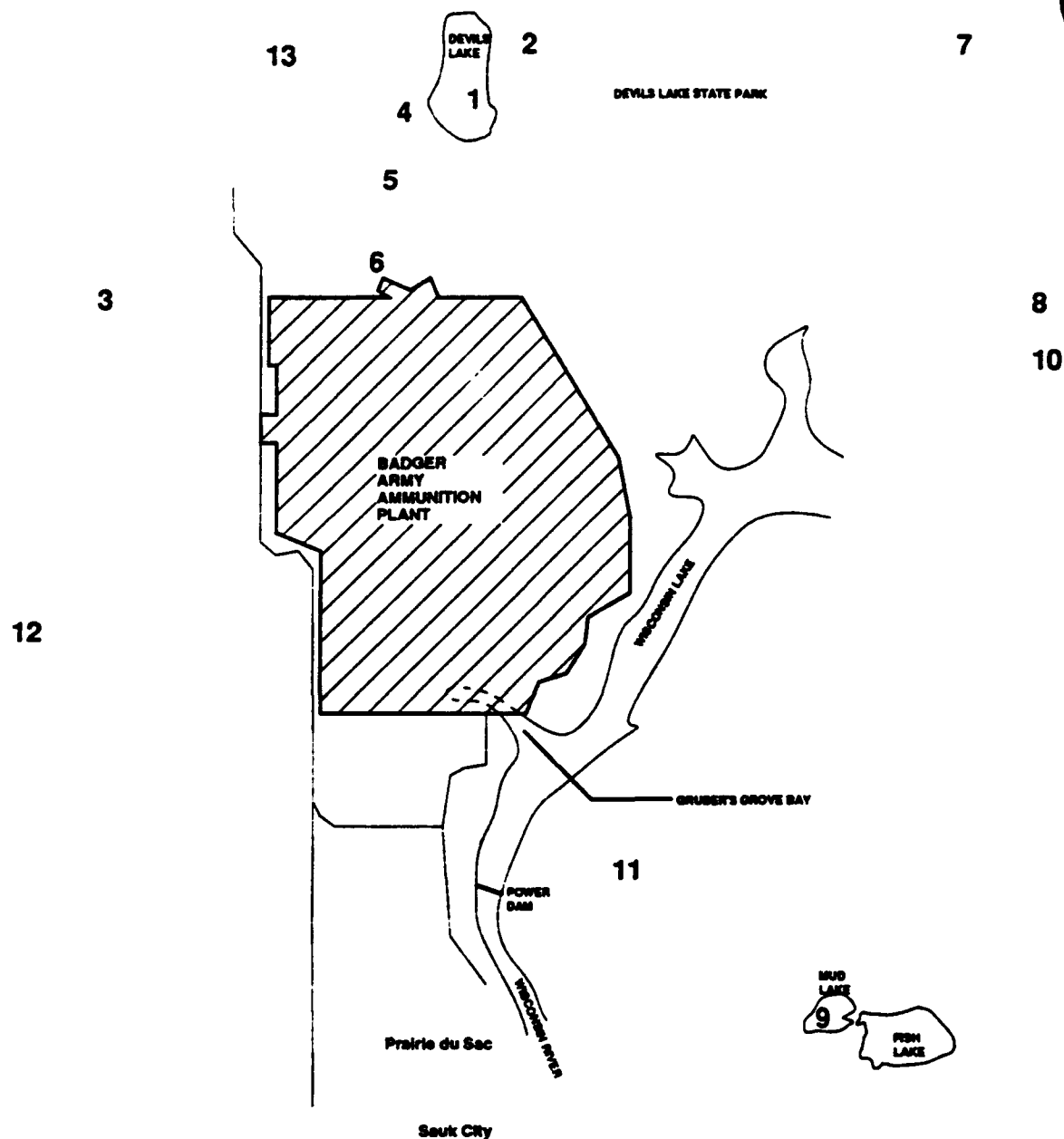


FIGURE P-1  
LOCATION OF HABITATS IN THE VICINITY  
OF BAAP THAT CONTAIN SPECIAL STATUS SPECIES  
REMEDIAL INVESTIGATION  
BADGER ARMY AMMUNITION PLANT

## KEY FOR FIGURE P-1

MAP POINT	LOCATION	DISTANCE FROM BAAP
1	Devil's Lake	4 mi N
2	Devil's Lake Oak Forest	4 mi N
3	Baxter's Hollow	2 mi WNW
4	Koshawago Springs	2 mi N
5	Pine Hollow Headwaters	1.5 mi N
6	Pine Glen	0.5 mi N
7	Parfrey's Glen	5 mi NE
8	Owl's Head Hill	6 mi E
9	Mud Lake	5 mi SE
10	Gibraltar Rock	6 mi E
11	Blackhawk's Lookout	3.5 mi SE
12	Otter Creek Bluff	6 mi radius beginning 1 mi SW
13	Ski Hi Orchard	2 mi NNW
	Wisconsin River	Varies
	Wisconsin River-lower	3.5-4.5 mi S
	Wisconsin River-Prairie Du Sac	5.5 mi S
	Wisconsin River-NW of Gruber's Grove Bay	2.5 mi SE
	Dunlap Hollow	10 mi SE (Not Shown)

## APPENDIX P

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All three of these species have been observed in Baxter's Hollow (1.5 miles west-north-west of BAAP) and the Hooded Warbler and Worm-Eating Warbler have also been located in Pine Glen, which borders the northern boundary of BAAP (see figure P-1). The small home range and specialized habitat requirements for nesting and foraging suggest that these birds are probably not summer residents at BAAP, which is characterized by relatively open habitat. However, given the close proximity of Pine Glen to BAAP, it is possible that these birds may occasionally wander onto the compound, or use the compound as a stop during seasonal migrations.

### Peregrine Falcon (*Falco peregrinus*)

The Peregrine Falcon breeds in arctic North America and winters from the eastern U.S. west to British Columbia and south to the northern portion of South America in mountainous terrain. It has become extinct in many areas of the U.S. and occurrences are rare, as evidenced by population densities, which range from a high density of 1 nesting pair per 2000 square miles to a low density of 1 nesting pair per 20,000 square miles. Peregrine Falcons require high, rocky cliffs for nesting; a nearby source of water is preferred as well. These falcons forage for small to large birds and occasionally mammals (DeGraaf and Rudis, 1986).

The Peregrine Falcon have been spotted at Devil's lake (2.5 mi. north of BAAP), Gibraltar rock (6 mi. east of BAAP), and Otter Creek Bluffs (1-6 mi. southwest of BAAP). Though BAAP does not provide an adequate nesting habitat for this falcon, it is possible that individual birds may forage for food in the general vicinity of BAAP.

## II. REPTILES AND AMPHIBIANS

### Blanding's Turtle (*Emydoidea blandingii*)

The Blanding's turtle occurs in localized populations in the U.S., with the most dense population in the Great Lakes states, particularly Michigan and Wisconsin (Conant, 1975). The Blanding's turtle occupies wet areas with muddy bottoms including bogs, marshes, swamps, and pond or lake inlets. The turtle does not normally venture further than 100 meters from wetlands, but will occasionally disperse overland. Prey preferences consist primarily of crustaceans and aquatic insects, although plant matter comprises a significant proportion of the diet, at least seasonally (DeGraaf and Rudis, 1986).

This turtle has been found in and around Mud Lake, which is located six miles south of BAAP. Due to the lack of swamp habitat at BAAP, it is unlikely that the Blanding's turtle would inhabit BAAP.

Ornate Box turtle (*Terrapene ornata*)

The range of the Ornate Box turtle extends through the southern and central U.S. plains states; southern Wisconsin forms the northernmost boundary of their distribution (Conant, 1975). These turtles inhabit treeless plains with grass and low brush cover, especially near waterways. The home range does not typically extend beyond a 275 foot radius around its burrow and food habits generally include insects and vegetation (Ernst and Barbour, 1972).

The ornate box turtle has been found several miles south of BAAP at Dunlap Hollow. The habitat differences between BAAP and the area around Dunlap Hollow indicate that it is unlikely the Ornate Box turtle would inhabit BAAP.

### III. PLANTS

Drooping Sedge (*Carex prasina*)

This species occurs from Maine south to Maryland and west to Ohio, Michigan and southern Wisconsin in moist thickets, meadows, and low woods (Britton and Brown, 1970; Fernald, 1970). This plant has been located in wet, thickly forested areas such as Baxter's Hollow, Koshawago Springs, and around Devil's Lake. The generally dry and open environment at BAAP is not anticipated to support populations of this wetland plant.

Spotted Pondweed (*Potamogeton pulcher*)

This aquatic species occurs in peaty ponds and pools from Massachusetts to Georgia and west (Britton and Brown, 1970; Fernald, 1970). Although Spotted Pondweed has been found in Baxter's Hollow, it is unlikely that this plant would grow in the relatively dry and open environment at BAAP.

New York Monkshood (*Aconitum noveboracense*)

N.Y. Monkshood is found in damp, wooded ravines and slopes across the northern United States (Britton and Brown, 1970; Fernald, 1970). This plant has been located in the higher terrain to the northeast of BAAP in and around Pafrey's Glen. However, it is unlikely that New York Monkshood would grow in the prairie environment that characterizes BAAP.

Slender Bush-clover (*Lespedeza virginica*)

Slender Bush-clover requires dry soils in open woods, thickets and barrens and is found in eastern North America from New Hampshire, Ontario and Minnesota, south to Texas

## APPENDIX P

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(Britton and Brown, 1970). Slender Bush-clover has been found to the north of BAAP in and around the Devil's Lake and the Devil's Lake Oak Forest area and at Pine Glen. The habitat requirements of this plant may be met by conditions found at BAAP.

### Nuttall's Prairie Parsley (*Polytaenia nuttallii*)

Nuttall's Prairie Parsley occurs in prairies and open woods throughout the central U.S. extending from Michigan and Wisconsin, west to Kansas, and south to Louisiana and Texas (Britton and Brown, 1970; Fernald, 1970). This plant has been found approximately 5 miles north of BAAP on an intermittently vegetated prairie on the north side of Devil's Lake. Based on proximity and habitat similarities it is possible that this plant may occur at the BAAP site.

### Gattinger's Agalinis (Round-stemmed False Fox Glove) (*Agalinus gattingeri*)

This species is found in dry, open woodlands and siliceous slopes from Wisconsin, west to Iowa, and south to Tennessee and Texas (Britton and Brown, 1970; Fernald, 1970). This plant has been located to the north and east of BAAP, including Devil's Lake and Devil's Lake Oak forest, Pafrey's Glen, and Pine Glen. Because Gattinger's Agalinis grows best in dry, open woodland and barrens, it is possible that it would grow in similar habitats at BAAP.

### Tubercled Orchis (*Plantanthera flava* var *herbiola*)

This species requires moist soil and grows from Nova Scotia to Minnesota and south to Louisiana and Missouri (Britton and Brown, 1970). The Tubercled Orchis has been found at Ski Hi Orchard and Pine Hollow Headwaters just to the northwest of the Pine Glen area. It requires moist soils, and most likely grows near water within the forested parts of these areas. Based on its preference for moist, wooded habitats, it is unlikely that the Tubercled Orchis would be found at BAAP.

### Purple Milkweed (*Asclepias purpurascens*)

Purple Milkweed is found in dry fields and thickets from New Hampshire to Minnesota, and south to Arkansas and North Carolina (Britton and Brown, 1970). This plant has been found in the Pine Glen area and might occur in the northern half of the BAAP site.

### Yellowish Gentian (*Gentiana alba*)

The Yellowish Gentian occurs in moist soils in the eastern and central U.S. including Minnesota to Virginia and Kentucky (Britton and Brown, 1970). Although this plant has



been found around the Blackhawk Ridge area, which is five miles south of BAAP and about one-half mile east of the Wisconsin river, it is unlikely to occur at BAAP.

Wooly Milkweed (*Asclepias lanuginosa*)

This species is found on prairies from N. Illinois to Minnesota and west to Wyoming (Britton and Brown, 1970). Wooly Milkweed occurs in the Owl's Head Hill area, six to seven miles east of BAAP and may be found in similar habitats at BAAP.

#### IV. FISH AND MUSSELS

Several endangered or threatened mussel and fish species have been located in various regions of the Wisconsin river. Fish species include Paddlefish (*Polyodon spathula*), Blue Sucker (*Cycleptus elongatus*), Black Buffalo (*Ictiobus niger*), Lake Sturgeon (*Acipenser fulvescens*), Goldeye (*Hiodon alosoides*), and Speckled Chub (*Macrhybopsis aestivalis*); mussels include Rock Pocketbook (*Arcidens confragosus*), Higgins' Eye (*Lampsilis higginsii*), Monkeyface (*Quadrula metanevra*), Paper Pondshell (*Anodonta imbecillis*), Round Pigtoe (*Pleurobema sintoxia*), Elktoe (*Alasmidonta marginata*), and Buckhorn (*Tritogonia verrucosa*).

#### V. WATCH LIST ELEMENTS

Several state watch list plant and animal species have also been located within an eight mile radius of BAAP. Plant species include: Hooker's Orchid (*Plantanthera hookeri*), Cliff Golden Rod (*Solidago sciaphila*), Vasey's Pond Weed (*Potamogeton vaseyi*), Poverty Grass (*Aristida dichotoma*), Large Water Starwort (*Callitriche heterophylla*), Dry Woods Sedge (*Carex aritecta*), Prairie Dandelion (*Nothocalais cuspidata*) Maidenhair's Spleenwort (*Asplenium trichomanes*), and Purple Cliff Brake (*Pellaea atropurea*). Insects include dragonflies (*Cordulegaster obliqua*, *Neurocordulia yamaskanensis*, and *Somatochlora tenebrosa*) and several species of butterflies including *Epargyreus clarus*, *Poanes massasoit* and *Euphyes conspicua*. In the Wisconsin river, the Pugnose minnow (*Opsopoeodus emiliae*) and Western Sand darter (*Ammocrypta Clara*) have been documented. The Wisconsin Watch List also contains a number of community types of interest known to occur within an eight mile radius of BAAP. These special habitats include: Southern dry-mesic forest, Southern dry forest, Northern dry-mesic forest, cliff and shaded cliff, open bog, and mesic prairie. In addition, mussel bed communities in the Wisconsin River have also been listed.

TABLE P-1  
NON-WOODY PLANT SPECIES OBSERVED OR REPORTED IN VICINITY OF BAAP [a]  
REMEDIAL INVESTIGATION  
BADGER ARMY AMMUNITION PLANT

SCIENTIFIC NAME	COMMON NAME
<i>Achillea millefolium</i>	Yarrow
<i>Arisaema triphyllum</i>	Jack-in-the-pulpit
<i>Asclepias syriaca</i>	Common milkweed
<i>Asclepias verticillata</i>	Whorled milkweed
<i>Asparagus sp.</i>	Asparagus
<i>Barbarea sp.</i>	Yellow rocket
<i>Bidens connata</i>	Beggars's tick
<i>Caltha sp.</i>	Cowslip
<i>Cannabis sp.</i>	Hemp
<i>Celastrus scandens</i>	Bittersweet
<i>Cerastium stellaria</i>	Chickweed
<i>Chenopodium sp.</i>	Pigweed
<i>Cichorium intybus</i>	Chicory
<i>Convolvulus sp.</i>	Bindweed
<i>Corylus cornuta</i>	Hazelbush
<i>Crataegus sp.</i>	Thorn apple
<i>Cynoglossum sp.</i>	Hound's tongue
<i>Daucus carota</i>	Queen Anne's lace
<i>Dicentra cucullaria</i>	Dutchman's breeches
<i>Diervilla sp.</i>	Bush honeysuckle
<i>Fragaria virginiana</i>	Wild strawberry
<i>Gaultheria procumbens</i>	Wintergreen
<i>Geranium sp.</i>	Wild geranium
<i>Geranium robertianum</i>	Herb robert
<i>Hemerocallis sp.</i>	Daylily
<i>Hieracium sp.</i>	Hawkweed
<i>Hypericum sp.</i>	St. Johns wort
<i>Impatiens capensis</i>	Jewelweed
<i>Iris versicolor</i>	Blue flag
<i>Lactuca sp.</i>	Wild lettuce
<i>Lathyrus sp.</i>	Vetch
<i>Lepidium sp.</i>	Pepper grass
<i>Linaria vulgaris</i>	Butter and eggs
<i>Lobelia cardinalis</i>	Cardinal flower, Red lobelia
<i>Medicago sativa</i>	Alfalfa
<i>Medicago lupinus</i>	Black medic
<i>Melilotus sp.</i>	Sweet clover
<i>Nepeta hederacea</i>	Creeping charlie
<i>Oenothera biennis</i>	Evening-primrose
<i>Philadelphus sp.</i>	Mock orange
<i>Phlox sp.</i>	Phlox
<i>Plantago major</i>	Common plantain

TABLE P-1  
NON-WOODY PLANT SPECIES OBSERVED OR REPORTED IN VICINITY OF BAAP [a]

REMEDIAL INVESTIGATION  
BADGER ARMY AMMUNITION PLANT

SCIENTIFIC NAME	COMMON NAME
<i>Podophyllum peltatum</i>	Mayapple
<i>Polygonatum sp.</i>	Solomons seal
<i>Polygonum cuspidatum</i>	Japanese knotweed
<i>Polygonum pennsylvanicum</i>	Pennsylvania smartweed
<i>Preridium aquilinum</i>	Bracken fern
<i>Rhus glabra</i>	Smooth sumac
<i>Rhus radicans</i>	Poison ivy
<i>Rubus allegheniensis</i>	Blackberry
<i>Rubus idaeus</i>	Red raspberry
<i>Rubus occidentalis</i>	Black raspberry
<i>Rudbeckia sp.</i>	Coneflower
<i>Sambucus canadensis</i>	Elderberry
<i>Smilax herbacea</i>	Carriion flower
<i>Syringa vulgaris</i>	Common lilac
<i>Taraxacum officinale</i>	Dandelion
<i>Tragopogon sp.</i>	Goatsbeard
<i>Trifolium sp.</i>	Clover
<i>Typha latifolia</i>	Cattail
<i>Verbascum thapsus</i>	Common mullein
<i>Verbena hastata</i>	Blue vervain
<i>Veronia sp.</i>	Ironweed
<i>Viola sp.</i>	Violet
<i>Vitis sp.</i>	Grape
<i>Xanthium sp.</i>	Cocklebur
<i>Zea mays</i>	Corn

Note:

a Based on Hellewell and Mattei, 1983

TABLE P-2  
TREE SPECIES OBSERVED OR REPORTED IN PROXIMITY TO BAAP [a]

REMEDIAL INVESTIGATION  
BADGER ARMY AMMUNITION PLANT

SCIENTIFIC NAME	COMMON NAME
<i>Acer negundo</i>	Box elder
<i>Acer nigrum</i>	Black maple
<i>Acer saccharum</i>	Sugar maple
<i>Betula sp.</i>	Birch
<i>Carya cordiformes</i>	Bitternut hickory
<i>Carya ovata</i>	Shagbark hickory
<i>Celtis occidentalis</i>	Hackberry
<i>Fraxinus americana</i>	White ash
<i>Juglans cinerea</i>	Butternut
<i>Juglans nigra</i>	Black walnut
<i>Juniperus virginiana</i>	Red cedar
<i>Morus rubra</i>	Red mulberry
<i>Picea glauca</i>	White spruce
<i>Pinus resinosa</i>	Red pine
<i>Pinus strobus</i>	White pine
<i>Populus deltoides</i>	Cottonwood
<i>Populus grandidentata</i>	Bigtooth aspen
<i>Prunus pensylvanica</i>	Pin cherry
<i>Prunus serotina</i>	Black cherry
<i>Prunus virginiana</i>	Choke cherry
<i>Pyrus malus</i>	Apple
<i>Quercus alba</i>	White oak
<i>Quercus macrocarpa</i>	Bur oak
<i>Quercus rubra</i>	Red oak
<i>Quercus velutina</i>	Black oak
<i>Robinia pseudoacacia</i>	Black locust
<i>Salix nigra</i>	Black willow
<i>Thuja occidentalis</i>	Northern white cedar
<i>Tilia americana</i>	American basswood
<i>Ulmus americana</i>	American elm
<i>Xanthoxylum americanum</i>	Prickly ash

Note:

a. Based on Hellewell and Mattei, 1983

TABLE P-3  
MAMMALS OBSERVED OR REPORTED IN PROXIMITY TO BAAP [a]  
REMEDIAL INVESTIGATION  
BADGER ARMY AMMUNITION PLANT

SCIENTIFIC NAME	COMMON NAME	HABITAT	RELATIVE ABUNDANCE
<i>Didelphis virginiana</i>	Virginia opossum	Woodlands	Abundant
<i>Glaucomys volans</i>	Southern flying squirrel	Woodlands	Rare
<i>Lasiurus borealis</i>	Red bat	Woodlands	Moderate
<i>Marmota monax</i>	Woodchuck	Woodland and Open Areas	Moderate
<i>Mephitis mephitis nigra</i>	Striped skunk	Woodland and Open Areas	Moderate
<i>Mus musculus</i>	House mouse	Buildings	Moderate
<i>Odocoileus virginianus borealis</i>	White-tailed deer	Woodlands	Abundant
<i>Ondatra zibethicus</i>	Muskrat	Ponds and Streams	Moderate
<i>Peromyscus maniculatus</i>	Deer mouse	Woodlands	Moderate
<i>Procyon lotor</i>	Raccoon	Woodlands	Abundant
<i>Scalopus aquaticus aquaticus</i>	Eastern mole	Underground	Moderate
<i>Sciurus carolinensis pennsylvanicus</i>	Gray squirrel	Woodlands	Moderate
<i>Sciurus niger</i>	Fox squirrel	Woodland and Open Areas	Abundant
<i>Sylvilagus floridanus</i>	Eastern cottontail rabbit	Woodland and Open Areas	Moderate
<i>Tamias striatus</i>	Eastern chipmunk	Open areas	Abundant
<i>Taxidea taxus</i>	Badger	Woodland and Open Areas	Moderate
<i>Urocyon cinereoargenteus</i>	Gray fox	Woodland and Open Areas	Rare
<i>Vulpes vulpes</i>	Red fox	Woodland and Open Areas	Moderate

Notes:

- Based on Hellewell and Mattel, 1983
- Sighted by ABB-ES field personnel

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TABLE P-4  
AVIFAUNA OBSERVED OR REPORTED IN PROXIMITY TO BAAP  
REMEDIAL INVESTIGATION  
BADGER ARMY AMMUNITION PLANT

	SCIENTIFIC NAME	COMMON NAME	RELATIVE ABUNDANCE
b, d	<i>Agelaius phoeniceus</i>	Red-winged blackbird	Moderate
h	<i>Aix sponsa</i>	Wood duck	Moderate
	<i>Anas acuta</i>	Northern pintail	Migratory
	<i>Anas discors</i>	Blue-winged teal	Moderate
b	<i>Anas platyrhynchos</i>	Mallard	Moderate
	<i>Archilochus colubris</i>	Ruby-throated hummingbird	Moderate
	<i>Ardea herodias</i>	Great blue heron	Rare
	<i>Aythya valisineria</i>	Canvasback	Migratory
	<i>Bombycilla cedrorum</i>	Cedar waxwing	Migratory
b	<i>Bonasa umbellus</i>	Ruffed grouse	Moderate
	<i>Branta canadensis</i>	Canada goose	Migratory
	<i>Bubo virginianus</i>	Great horned owl	Rare
b, c	<i>Buteo jamaicensis</i>	Red-tailed hawk	Moderate
b, c, d	<i>Cardinalis cardinalis</i>	Northern cardinal	Moderate
	<i>Carduelis tristis</i>	American goldfinch	Moderate
	<i>Carduelis purpureus</i>	Purple finch	Moderate
b	<i>Cathartes aura</i>	Turkey vulture	Moderate
c	<i>Centurus carolinus</i>	Red-bellied woodpecker	Moderate
	<i>Certhia americana</i>	Brown creeper	Moderate
b	<i>Charadrius vociferus</i>	Killdeer	Abundant
	<i>Chordeiles minor</i>	Common nighthawk	Moderate
c	<i>Coccyzus americanus</i>	Yellow-bellied cuckoo	Moderate
b	<i>Colaptes auratus</i>	Northern flicker	Moderate
b, d	<i>Colinus virginianus</i>	Northern bobwhite quail	Rare-Moderate
c	<i>Contopus virens</i>	Eastern pewee	Moderate
b, c, d	<i>Corvus brachyrhynchos</i>	American crow	Abundant
b, c, d	<i>Cyanocitta cristata</i>	Blue jay	Abundant
c	<i>Dendrocopos villosus</i>	Hairy woodpecker	Moderate
c	<i>Dendroica pensylvanica</i>	Chestnut-sided warbler	Migratory
	<i>Dolichonyx oryzivorus</i>	Bobolink	Abundant
c	<i>Dryocopus pileatus</i>	Pileated woodpecker	Moderate
c	<i>Dumetella carolinensis</i>	Gray catbird	Moderate
c	<i>Empidonax minimus</i>	Least flycatcher	Moderate
	<i>Eremophila alpestris</i>	Horned lark	Moderate
	<i>Euphagus cyanocephalus</i>	Brewer's blackbird	Moderate
b	<i>Falco sparverius</i>	American kestrel	Moderate
	<i>Hesperiphona vespertina</i>	Evening grosbeak	Moderate
h	<i>Hirundo rustica</i>	Barn swallow	Moderate

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TABLE P-4  
 AVIFAUNA OBSERVED OR REPORTED IN PROXIMITY TO BAAP  
 REMEDIAL INVESTIGATION  
 BADGER ARMY AMMUNITION PLANT

	SCIENTIFIC NAME	COMMON NAME	RELATIVE ABUNDANCE
	<i>Hylocichla mustelina</i>	Wood thrush	Moderate
	<i>Icterus galbula</i>	Northern oriole	Moderate
	<i>Iridoprocne bicolor</i>	Tree swallow	Moderate
d	<i>Junco hyemalis</i>	Junco	Moderate
c	<i>Melanerpes erythrocephalus</i>	Red-headed woodpecker	Rare
d	<i>Melospiza melodia</i>	Song sparrow	Moderate
	<i>Mniotilta varia</i>	Black-and-white warbler	Moderate
b,c	<i>Molothrus ater</i>	Brown-headed cowbird	Moderate
c	<i>Myiarchus crinitus</i>	Crested flycatcher	Moderate
c	<i>Oporornis philadelphia</i>	Mourning warbler	Migratory
b,c,d	<i>Parus atricapillus</i>	Black-capped chickadee	Moderate
	<i>Parus bicolor</i>	Tufted titmouse	Moderate
b	<i>Passer domesticus</i>	House sparrow	Moderate
d	<i>Passerella iliaca</i>	Fox sparrow	Moderate
c	<i>Passerina cyanea</i>	Indigo bunting	Moderate
b	<i>Phasianus colchicus</i>	Ring-necked pheasant	Moderate
c	<i>Pheucticus ludovicianus</i>	Rose-breasted grosbeak	Rare
b,c,d	<i>Picoides pubescens</i>	Downy woodpecker	Moderate
	<i>Pipilo erythrophthalmus</i>	Rufous-sided towhee	Rare
c	<i>Piranga olivacea</i>	Scarlet tanager	Rare
d	<i>Poocetes gramineus</i>	Vesper sparrow	Moderate
c	<i>Progne subis</i>	Purple martin	Moderate
	<i>Regulus calendula</i>	Ruby-crowned kinglet	Moderate
	<i>Regulus satrapa</i>	Golden-crowned kinglet	Moderate
	<i>Scolopax minor</i>	American woodcock	Migratory
c	<i>Sciurus aurocapillus</i>	Oven bird	Moderate
c	<i>Setophaga ruticilla</i>	American redstart	Moderate
b	<i>Sialia sialis</i>	Eastern bluebird	Rare
b,c	<i>Sitta carolinensis</i>	White-breasted nuthatch	Moderate
	<i>Sphyrapicus varius</i>	Yellow-bellied sapsucker	Moderate
c	<i>Spinus tristis</i>	American goldfinch	Moderate
d	<i>Spizella passerina</i>	Chipping sparrow	Moderate
c	<i>Strix varia</i>	Barred owl	Moderate
b	<i>Sturnella magna</i>	Eastern meadowlark	Abundant
b	<i>Sturnus vulgaris</i>	European starling	Moderate
	<i>Toxostoma rufum</i>	Brown thrasher	Moderate
b,c	<i>Troglodytes aedon</i>	House wren	Abundant
b,c	<i>Turdus migratorius</i>	American robin	Abundant

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TABLE P-4  
 AVIFAUNA OBSERVED OR REPORTED IN PROXIMITY TO BAAP  
 REMEDIAL INVESTIGATION  
 BADGER ARMY AMMUNITION PLANT

	SCIENTIFIC NAME	COMMON NAME	RELATIVE ABUNDANCE
	<i>Tyrannus tyrannus</i>	Eastern kingbird	Moderate
c	<i>Vireo olivaceus</i>	Red-eyed vireo	Moderate
c	<i>Wilsonia citrina</i>	Hooded warbler	Migratory
b,d	<i>Zenaida macroura</i>	Mourning dove	Abundant

Notes:

- a. Based on Hellewell and Mattei, 1983.
- b. Sighted by ABB-ES field personnel, 1989.
- c. Mossman and Lange, 1982 (survey sites number 10 and 65; Eschenbach Oak Woods [T11N R7E S.24 SE, 25SW] and South Bluff Oak Forest [T11N R7E S.31], respectively).
- d. Wegner, 1985



TABLE P-5  
FISH OBSERVED OR REPORTED IN PROXIMITY TO BAAP (a)  
REMEDIAL INVESTIGATION  
BADGER ARMY AMMUNITION PLANT

SCIENTIFIC NAME	COMMON NAME	HABITAT
<i>Acipenser fulvescens</i>	Lake sturgeon	Lakes, Rivers
<i>Esox lucius</i>	Northern pike	Lakes, Rivers
<i>Ictalurus nebulosus</i>	Brown bullhead	Ponds
<i>Lepomis macrochirus</i>	Bluegill	Ponds, Lakes
<i>Lepomis</i> sp.	Sunfish	Ponds, Lakes
<i>Micropterus dolomieu</i>	Smallmouth bass	Ponds, Lakes
<i>Perca flavescens</i>	Yellow perch	Ponds, Streams
<i>Pimephales promelas</i>	Fathead minnow	Ponds, Streams
<i>Pomoxis</i> sp.	Crappie	Ponds, Streams, Rivers
<i>Roccus chrysops</i>	White bass	Ponds, Streams, Rivers
<i>Stizostedion vitreum vitreum</i>	Walleye	Lakes, Rivers

Note:

a. Based on Data Reported in EIS for Wastewater Treatment Facility, City of Portage, Wisconsin (WDNR, 1981) and Hellewell and Mattei, 1983.

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## APPENDIX P

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TABLE P-6  
THREATENED AND ENDANGERED SPECIES

ORGANISM	LOCATION	POTENTIAL OCCURRENCE AT BAAP
PLANTS		
New York Monkshood <sup>1</sup>	Pafrey's Glen	NO
Slender Bush-Clover <sup>1</sup>	Devil's Lake Oak Forest	POSSIBLE
	Devil's Lake, Pine Glen	
Gattinger's Agalinis <sup>1</sup>	Devil's Lake Oak Forest	POSSIBLE
	Devil's Lake, Pine Glen	
	Pafrey's Glen	
Spotted Pond Weed <sup>2</sup>	Baxter's Hollow	NO
Tubercled Orchid <sup>1</sup>	Ski Hi Orchard	NO
	Pine Hollow Headwaters	
Drooping Sedge <sup>1</sup>	Baxter's Hollow, Devil's Lake	NO
	Koshawago Springs	
Purple Milkweed <sup>2</sup>	Pine Glen	POSSIBLE
Wooly Milkweed <sup>1</sup>	Owl's Head Hill	POSSIBLE
Yellowish Gentian <sup>1</sup>	Blackhawk's Lookout	NO
Nuttall's Prairie Parsley <sup>1</sup>	North of Devil's Lake	POSSIBLE
ANIMALS		
BIRDS		
Kentucky Warbler <sup>1</sup>	Baxter's Hollow	POSSIBLE
Hooded Warbler <sup>1</sup>	Baxter's Hollow, Pine Glen	POSSIBLE
Worm-eating Warbler <sup>2</sup>	Baxter's Hollow, Pine Glen	POSSIBLE
Cooper's Hawk <sup>3</sup>	Mud Lake, Pine Glen	POSSIBLE
Peregrine Falcon <sup>2</sup>	Devil's Lake, Gibraltar Rock, Otter Creek Bluff	POSSIBLE
REPTILES		
Blanding's Turtle <sup>1</sup>	Mud Lake	NO
Ornate Box Turtle <sup>2</sup>	Dunlap Hollow	NO

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**APPENDIX P**

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**FISH**

Paddlefish <sup>1</sup>	WI River, WI River-Prairie Du Sac	NO
Blue Sucker <sup>1</sup>	WI River	NO
Black Buffalo <sup>1</sup>	WI River	NO
Lake Sturgeon <sup>3</sup>	WI River	NO
Goldeye <sup>2</sup>	WI River	NO
Speckled Chub <sup>1</sup>	WI River	NO

**MUSSELS**

Paper Pondshell <sup>3</sup>	WI River-lower	NO
Round Pigtoe <sup>3</sup>	WI River-lower	NO
Rock Pocketbook <sup>1</sup>	WI River-lower	NO
Buckhorn <sup>1</sup>	WI River-lower	NO
Elktoe <sup>3</sup>	WI River-lower	NO
Higgins' Eye <sup>2</sup>	WI River-lower	NO
Monkeyface <sup>1</sup>	WI River-lower	NO
Winged Mappleleaf <sup>2</sup>	Baraboo River	NO

1: Threatened species

2: Endangered species

3: Federally protected species.

## APPENDIX P

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### STATE OF WISCONSIN WATCH LIST SPECIES

#### COMMUNITY HABITATS:

Southern dry-mesic forest  
Southern dry forest  
Northern dry-mesic forest  
cliff and shaded cliff  
open bog  
mesic prairie  
mussel bed

#### PLANTS:

Hookers orchid: Pafrey's Glen  
Cliff Goldenrod: Black Hawk's Lookout and Owl's head hill  
Vasey's Pond Weed, Poverty grass, Large water starwort, dry woods sedge, Maidenhair  
Spleenwort: Devil's lake and Pine Glen  
Purple Cliff Brake: Owl's Head Hill

#### ANIMALS:

Dragonfly (arrow head spiketail): Baxter's hollow  
Butterflies (several species): Baxter's hollow, Devil's lake  
Fish (western sand darter (*Amocrypta clara*), Pugnose Minnow (*Opsopoeodus emiliae*):  
Wisconsin river

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REMEDIAL INVESTIGATION  
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**APPENDIX Q**  
**EXPOSURE PARAMETERS OF SITE SPECIES**

TABLE Q-1  
BIOACCUMULATION FACTORS FOR EXPOSURE MODELING  
REMEDIAL INVESTIGATION  
BADGER ARMY AMMUNITION PLANT

BIOACCUMULATION FACTORS [a]						
CHEMICAL	log Kow	PLANT	[b] INVERTS	SMALL MAMMAL	SMALL BIRD	HERPTILE
VOLATILE ORGANICS						
ACET	-0.24	1	1	1	1	1
C6H6	2.15	1	1	1	1	1
MEK	0.29	1	1	1	1	1
CH2CL2	1.54	1	1	1	1	1
SEMI-VOLATILE ORGANICS						
ANAPNE	4	0.189	1	1	1	1
ANAPYL	3.93	0.207	1	1	1	1
ANTRC	4.45	0.104	1	1	1	1
B2EHP	5.11	0.043	1	1	1	1
BAANTR	5.6	0.022	1	1	1	1
BAPYR	6.06	0.012	1	1	1	1
BBFANT	6.06	0.012	1	1	1	1
BKFANT	6.06	0.012	1	1	1	1
BGHIPIY	6.51	0.007	1	1	1	1
CHRY	5.61	0.022	1	1	1	1
DBAHA	6.8	0.005	1	1	1	1
DEP	3.22	0.533	1	1	1	1
DNBP	4.80	0.065	1	1	1	1
24DNT	1.98	1	1	1	1	1
26DNT	1.72	1	1	1	1	1
DPA	--	1	1	1	1	1
FANT	4.9	0.057	1	1	1	1
FLRENE	4.9	0.057	1	1	1	1
ICDPYR	6.5	0.007	1	1	1	1
2MNAP	7.94	0.001	1	1	1	1
NAP	3.37	0.437	1	1	1	1
NC	--	0.05	0.05	0	0	0
NG	2.04 [m]	1	1	1	1	1
NNDPA	3.13	0.597	1	1	1	1
NNDMEA	-.57	1	1	1	1	1
NNDNPA	1.36	1	1	1	1	1
PHANTR	4.46	0.102	1	1	1	1
123PDA	--	1	1	1	1	1
PYR	4.88	0.059	1	1	1	1
CCL3F	2.53	1	1	1	1	1

**TABLE Q-1**  
**BIOACCUMULATION FACTORS FOR EXPOSURE MODELING**  
**REMEDIAL INVESTIGATION**  
**BADGER ARMY AMMUNITION PLANT**

CHEMICAL	log Kow	BIOACCUMULATION FACTORS [a]				
		PLANT	[b] INVERTS	SMALL MAMMAL	SMALL BIRD	HERPTILE
INORGANIC COMPOUNDS						
AL	--	1	1	1	1	1
NH3	--	0.05	0.05	0	0	0
SB	--	1	1	1	1	1
AS	--	0.2 [d]	1	0.37 [e]	0.56 [f]	1
BA	--	1	1	1	1	1
BE	--	1	1	1	1	1
BR	--	0.05	0.05	0	0	0
CD	--	15.0 [g]	17 [h]	2.61 [h]	10 [g]	10 [g]
CL	--	0.05	0.05	0	0	0
CR	--	0.1 [i]	0.16 [c]	1	1	1
CU	--	10.0 [g]	9.25 [h]	1	1	1
PB	--	0.2 [j]	2.43 [h]	0.43 [h]	0.38 [j]	1
HG	--	1	0.34 [k]	5 [k]	2.33 [k]	10 [g]
NI	--	3.20 [l]	1.85 [h]	0.12 [l]	1	1
NIT	--	0.05	0.05	0	0	0
SE	--	1	1	1	1	1
AG	--	1	1	1	1	1
SO4	--	0.05	0.05	0	0	0
SN	--	1	1	1	1	1
ZN	--	10 [g]	7.31 [h]	5.11 [h]	10 [g]	10 [g]



**TABLE Q-1**  
**BIOACCUMULATION FACTORS FOR EXPOSURE MODELING**  
**REMEDIAL INVESTIGATION**  
**BADGER ARMY AMMUNITION PLANT**

CHEMICAL	log K <sub>ow</sub>	BIOACCUMULATION FACTORS [a]			
		PLANT	[b] INVERTS	SMALL MAMMAL	SMALL BIRD    HERPTILE

**NOTES:**

- [a] Bioaccumulation Factors (BAFs) were typically estimated to be 1 when empirical data were unavailable. However, plant and invertebrate BAFs were assumed to be 0.05, and vertebrate BAFs assumed to be 0 for those constituents without known tendency to bioaccumulate.  
 Plant BAFs for organic compounds were set equal to 1 when equation presented in [b] exceeded 1.
- [b] Calculated using the following equation in USEPA (1990):  
 $\log(\text{Plant Uptake Factor}) = 1.588 - 0.578 \log K_{ow}$
- [c] Assumption
- [d] Plant value from Eisler, 1988.
- [e] Mammal value from USEPA, 1985
- [f] Bird value from USEPA, 1985.
- [g] Conservative BAF estimate.
- [h] Values for earthworms and small mammals from McFadyen, 1980.
- [i] Plant value from USEPA, 1985c.
- [j] Earthworm and chicken value from USEPA, 1985d.
- [k] Invertebrate, mammal, and bird value from USEPA, 1985e.
- [l] Plant and small mammal value from USEPA, 1985f.
- [m] Log K oil-water

TABLE Q-2  
EXPOSURE PARAMETERS FOR INDICATOR TERRESTRIAL SPECIES  
REMEDIAL INVESTIGATION  
BADGER ARMY AMMUNITION PLANT

RECEPTOR SPECIES	EXPOSURE PARAMETER	REPORTED VALUES	REFERENCE	VALUE SELECTED FOR ECOLOGICAL RISK
Eastern Garter Snake ( <i>Thamnophis s. sirtalis</i> )	Home Range (acres)	5, 2, 35 (males), 22.2 (females)	DeGraaf and Rudis, 1986	5 [a]
	Percent Prey Items	Earthworms are 80% of diet; rest is amphibians, carrion, fish, leeches, caterpillars, insects, small birds, rodents, slugs, snakes, mollusks, crayfish, and sowbugs	DeGraaf and Rudis, 1986	Invertebrates: 85% Small Mammals: 5% Birds: 5% Soil: 5%
	Ingestion Rate (kg/day)	Allometric relationship between body weight (W) and food ingestion rate (F) for all species $F = 0.065 \times W^{0.7919}$	USEPA, 1988	0.023 kg/day
	Body Weight (kg)			0.27 kg [b]
	Drinking Water Intake Rate (l/day)	Allometric relationship between body weight (W) and drinking water rate (L) for all species $L = 0.11 \times W^{0.7872}$	USEPA, 1988	0.039 l/day

[a] Selected as conservative value; actual range may be greater.

[b] Estimated assuming the density of water (1 gm/cu.cm), an average length of 55 cm (Conant, 1975), and an assumed diameter of 2.5 cm.

**TABLE Q-2**  
**EXPOSURE PARAMETERS FOR INDICATOR TERRESTRIAL SPECIES**  
**REMEDIAL INVESTIGATION**  
**BADGER ARMY AMMUNITION PLANT**

RECEPTOR SPECIES	EXPOSURE PARAMETER	REPORTED VALUES	REFERENCE	VALUE SELECTED FOR ECOLOGICAL RISK																		
Red Fox ( <i>Vulpes vulpes</i> )	Home Range (acres)	< 3 miles in diameter; 142 – 400 Ac < 5 miles in diam.	DeGraaf and Rudis, 1986 Godin, 1977	250 [a]																		
	Percent Prey Items	142 to 1280; 900; 1495; 955 acres Birds, turtles, frogs, snakes, eggs, snowshoe hare, deer, porcupine, and berries and fruit when available	Baker, 1983 DeGraaf and Rudis, 1986	Invertebrates: 20% Plants: 10% Small Mammals: 40% Herpetofauna: 15% Birds: 10% Soil: 5%																		
		Small mammals, birds and their eggs, insects, earthworms, turtles and their eggs, frogs, snakes, wild berries, sarsaparilla, grapes, plums, and apples. Infrequently eats nuts and grains, and sometimes ingests rope, twine, paper, sticks, and trash.	Godin, 1977																			
		Mice, rabbits, other small mammals and birds, insects, carrion, fleshy fruits, and seeds. The percentage of plant material in diet varies seasonally as shown below:	Martin, et al., 1951																			
		<table><tr><th>Season</th><th>No. Month</th><th>Percent</th></tr><tr><td>Winter</td><td>5</td><td>4%</td></tr><tr><td>Spring</td><td>2</td><td>0%</td></tr><tr><td>Summer</td><td>3</td><td>31%</td></tr><tr><td>Fall</td><td>2</td><td>23%</td></tr><tr><td>Estimated Year – round Average</td><td></td><td>13%</td></tr></table>	Season	No. Month	Percent	Winter	5	4%	Spring	2	0%	Summer	3	31%	Fall	2	23%	Estimated Year – round Average		13%		
	Season	No. Month	Percent																			
	Winter	5	4%																			
	Spring	2	0%																			
	Summer	3	31%																			
	Fall	2	23%																			
Estimated Year – round Average		13%																				
Ingestion Rate (kg/day)	Allometric relationship between body weight (W) and food ingestion rate (F) for all species: $F = 0.065 \times W^{0.7919}$		USEPA, 1988	0.23 kg/day																		
Body Weight (kg)	3.6 to 5.4 kg 3.6 to 6.8 kg		Godin, 1977 Baker, 1983	4.9 [b]																		
Drinking Water Intake Rate (l/day)	Allometric relationship between body weight (W) and drinking water intake rate (L) for all species: $L = 0.11 \times W^{0.7872}$		USEPA, 1988	0.38 l/day																		

[a] Selected as conservative value; actual range may be much greater

[b] Average of reported values

TABLE Q-2  
EXPOSURE PARAMETERS FOR INDICATOR TERRESTRIAL SPECIES  
REMEDIAL INVESTIGATION  
BADGER ARMY AMMUNITION PLANT

RECEPTOR SPECIES	EXPOSURE PARAMETER	REPORTED VALUES	REFERENCE	VALUE SELECTED FOR ECOLOGICAL RISK
Short-tailed Shrew ( <i>Blarina brevicauda</i> )	Home Range (acres)	2.88, 1.0, 2.1, 1.46, 1.39, 0.25, 4.43 1.1, 25, 0.5, 1 0.5	Baker, 1983 DeGraaf and Rudis, 1986 Burt, 1987	1.3 [a]
	Percent Prey Items	Insects, invertebrates, small vertebrates, worms	Baker, 1983	Invertebrates: 85% Plants: 10% Soil: 5%
		Insects, plants, worms, sowbugs, snails, small vertebrates, centipedes, millipedes, spiders	DeGraaf and Rudis, 1986	
		Insects, earthworms, vertebrates, invertebrates, occasionally plants	Godin, 1977	
	Ingestion Rate (kg/day)	50% to 300% of its body weight/day	Baker, 1983	0.037 kg/day (175% of BW/day [a])
	Body Weight (kg)	0.018 to 0.030 kg	Baker, 1983	0.021 kg [a]
	Drinking Water Intake Rate (l/day)	0.013 to 0.024 kg	Godin, 1977	
		Allometric relationship between body weight (W) and drinking water rate (L) for mammals: $L = 0.10 \times W^{0.7377}$	USEPA, 1988	0.0058 l/day

[a] Average of reported values

**TABLE Q-2**  
**EXPOSURE PARAMETERS FOR INDICATOR TERRESTRIAL SPECIES**  
**REMEDIAL INVESTIGATION**  
**BADGER ARMY AMMUNITION PLANT**

RECEPTOR SPECIES	EXPOSURE PARAMETER	REPORTED VALUES	REFERENCE	VALUE SELECTED FOR ECOLOGICAL RISK
Red-tailed Hawk ( <i>Buteo jamaicensis</i> )	Home Range (acres)	Breeding: 192 - 1376 acres Winter: up to 2560 acres	DeGraaf and Rudis, 1986	500 [a]
	Percent Prey Items	Small mammals, amphibians, reptiles, nesting birds, insects, carrion, domestic animals	DeGraaf and Rudis, 1986	Small Mammals: 55% Invertebrates: 5% Plants: 5% Birds: 20% Herpetofauna: 10% Soil: 5%
	Ingestion Rate (kg/day)		Terres, 1987	0.23 kg/day [b]
	Body Weight (kg)	1.5 kg	Terres, 1987	1.5
	Drinking Water Intake Rate (l/day)	Allometric relationship (all species) $L = 0.11 \times W^{0.7872}$ $W = \text{Weight} = 1.50 \text{ kg}$	USEPA, 1988	0.151 l/day
	Density (#/acre)	0.0014 (1 pair/2.2 square miles) 0.00076 (1 pair/4.1 square miles) 0.00625 (1 pair/0.5 square miles)	DeGraaf and Rudis, 1986	0.0028 [c]
	Lifespan (years)	4 years	Terres, 1987	4

[a] Selected as conservative value; actual range may be much greater  
[b] Ingestion rate based upon ratio of ingestion rate to body weight for golden eagle (Terres, 1987) using 1.5 kg body weight for hawk  
[c] Average of reported values

**TABLE Q-2**  
**EXPOSURE PARAMETERS FOR INDICATOR TERRESTRIAL SPECIES**  
**REMEDIAL INVESTIGATION**  
**BADGER ARMY AMMUNITION PLANT**

RECEPTOR SPECIES	EXPOSURE PARAMETER	REPORTED VALUES	REFERENCE	VALUE SELECTED FOR ECOLOGICAL RISK
Eastern Meadowlark ( <i>Sturnella magna</i> )	Home Range (acres)	2.8 acres 7.0 acres Mean = 5	DeGraaf and Rudin, 1986 Terres, 1987	5 Acres
	Food Habits	Major foods include insects, weed seeds, & grass seeds.  74% of diet is invertebrates, including beetles, grubs, bugs, grasshoppers, crickets, ants, and spiders.	DeGraaf and Rudin, 1986  Terres, 1987	Invertebrates: 75% Plants: 20% Soil: 5%
	Body Weight (kg)	Western Meadowlark (87.5 g)	Terres, 1987	0.087 kg
	Drinking Water Intake Rate (l/day)	Allometric relationship between body weight (W) and drinking water rate (L) for chickens: $L = 0.13 \times W^{0.7555}$	USEPA, 1988	0.02 l/day
	Ingestion Rate (kg/day)	Allometric relationship between body weight (W) and food ingestion rate (F) for chickens: $F = 0.075 \times W^{0.8449}$	USEPA, 1988	0.0095 kg/day

**TABLE Q-3**  
**APPLICABLE SURFACE WATER AND SEDIMENT CRITERIA FOR AQUATIC ORGANISMS**

**REMEDIAL INVESTIGATION**  
**BADGER ARMY AMMUNITION PLANT**

COMPOUND	SURFACE WATER					RTV (d)	
	FEDERAL (EPA) WQC ACUTE (a) ( $\mu\text{g/L}$ )	CHRONIC ( $\mu\text{g/L}$ )	WISCONSIN WQC ACUTE (b) ( $\mu\text{g/L}$ )	CHRONIC ( $\mu\text{g/L}$ )	WISCONSIN WADV (c) ( $\mu\text{g/L}$ )	Surface Water ( $\mu\text{g/L}$ )	Sediment ( $\mu\text{g/g}$ )
AL	750 (e)	87 (e)	-	-	2,940	748	-
NH3							75 (n)
AS	360 (f)	190 (f)	363.8	153	171	153	
BA	13,600 (g,h)	13,600 (g,i)	-	-	11,500	1,360	
BE	130	5.3	-	-	32.6	5.3	
CL	860,000	230,000	-	-	94,300	94,300	
CR	16	11	14.2	9.74	3,710	9.74	100 (o)
CU	18 (i)	12 (i)	16.58 (i)	11.51 (i)	2.27	2.27	
FE		1,000 (g)				1,000	
PB	82 (i)	3.2 (i)	10.09 (i)	10.09 (i)	61.4	3.2	50 (o)
MN	1,000 (g)	100 (i)	-	-	128,000	100	
HG	2.4	0.012	1.53	-	0.028	0.012	0.1 (o)
NI	1,400 (i)	160 (i)	1,078 (i)	66.13 (i)	1,710	66.13	
NIT	5,000 (j)	5,000 (j)	-	-	1,250,000	5,000	545 (p)
CO4	-	-	-	-	1,060,000	1,060,000	-
EN	1,600 (k)	200 (k)	-	-	1,010	200	
	120 (i)	110 (i)	112.8 (i)	49.59 (i)	6,030	49.59	
B2EHP							-
PHANTR							1,390 (q)
NH3N2	-	2,100 (m)	-	-		2,100	

**Notes:**

- (a) Values are acute or chronic USEPA Ambient Water Quality Criteria or Lowest Observed Effect Levels unless otherwise indicated.
- (b) Values from Wisconsin Water Quality Standards (WAC, Chapter NR 105).
- (c) Wisconsin Wildlife and Domestic Values; calculated as described in WAC, Chapter NR 105.
- (d) Reference Toxicity Value (RTV); for surface water: derived as lowest of the federal and state chronic WQC and the state WDAV. Sediment RTVs are based on sediment criteria, guidelines, or effect threshold levels.
- (e) pH-dependent AWQC (pH=7.0 assumed).
- (f) Chronic AWQC for trivalent arsenic (USEPA 1984).
- (g) Water Quality Handbook (USEPA 1976).
- (h) Calculated by applying a factor of 0.2 to the acute LC50; this value is expected to protect 99.9% of the exposed population from acute effects (USEPA 1986).
- (i) Hardness-dependent criteria (100 mg/L CaCO3 assumed).
- (j) No Observed Adverse Effect Level (NOAEL for warmwater fish (USEPA 1986a).
- (k) Proposed water quality criteria for zebrafish (Beusen and Neven 1987).
- (l) Estimated by applying an acute-chronic ratio of 10.
- (m) Temperature and pH-dependent criteria for total NH3 (assumed pH=7.0 @ 20C).
- (n) Nonpolluted guideline for classifying sediments of Great Lakes Harbors (Anon 1977).
- (o) Wisconsin Department of Natural Resources Sediment Quality Criteria (Sullivan et al., 1985).
- (p) No effect level proposed by Ontario Ministry of Environment (Persaud et al., 1989).
- (q) USEPA interim mean freshwater sediment quality criterion, assuming 1% total organic carbon.
- No toxicological information for this compound is available.

**TABLE Q-4**  
**SUMMARY OF INGESTION TOXICITY DATA FOR TERRESTRIAL WILDLIFE**  
**REMEDIAL INVESTIGATION**  
**BADGER ARMY AMMUNITION PLANT**

CHEMICAL	TEST SPECIES	TEST TYPE	DURATION	EFFECT	ACUTE*			CHRONIC*		
					ORAL LD50 (mg/kg BW)	ACUTE ORAL RISK CRITERIA (mg/kg BW)	LOAEL (mg/kg BW/day)	NOAEL (mg/kg BW/day)	REFERENCE	
VOLATILE ORGANICS										
ACET	Rat	Single oral dose		Mortality	9750	1950 [a]				Sax, 1984
	Rat	Oral (subchronic)		Increased liver/kidney weight			500 [d]	100	IRIS, 1991	
MEK	Rat	Inhalation	NS	Fetotoxicity		1305	131 [b]		IRIS, 1991	
C6H6	Rat	Single oral dose		Mortality	3800	760 [a]	76 [b]		TDB, 1984	
	Rat	Oral (chronic)	187 days	Hematopoietic effects		100 [b]	10	1	USEPA, 1984b	
CH2CL2	Rat	Oral (subchronic)	3 months	Mortality, blood chemistry, histopathology				12.5	USEPA, 1984d	
	Rat	Oral (chronic)	2 years	Liver toxicity		526 [b]	52.6	5.9	IRIS, 1991	
SEMIVOLATILE ORGANICS										
ANAPNE	Mouse	Oral (chronic)	90 days	Hepatotoxicity		3500 [b]	350	175	IRIS, 1990	
	Rat	Oral (chronic)	32 days	Physiological changes		2000 [b]	2000		USEPA, 1984a	
ANAPYL	Rat	Oral (chronic)	40 days	Physiological changes		6000 [b]	600		USEPA, 1984a	
ANTRC	Rodents	Oral (chronic)	NS	Carcinogenicity		33000 [b]	3300		Eisler, 1987	
	Mouse	Oral (chronic)	90 days	No effects				1000	IRIS, 1990	
B2EHP	Rat	Single oral dose		Mortality	8600	1720 [a]	172 [b]		NIOSH, 1985	
	Rat	Single oral dose		Mortality	26000	5200 [a]	520 [b]		ATSDR, 1988	
	Guinea pig	Oral (chronic)	1 year	Increased liver weight			19		IRIS, 1992	
BAANTR	Rodents	Oral (chronic)	NS	Carcinogenicity		20 [b]	2		Eisler, 1987	
PAFTR	Rat	Oral (chronic)	Pregnancy	Sterility in offspring					USEPA, 1984c	
	Rodents	Oral (chronic)	NS	Carcinogenicity		0.02 [b]	40		Eisler, 1987	
	Rat	Oral (chronic)	NS	Papillomas in stomach			0.002		USEPA, 1984a	
	Rat	Oral (chronic)	Pregnancy	Decreased gonad weight			2.5		USEPA, 1984c	
	Rat	Oral (chronic)	3.5 months	Reproductive effects			10		USEPA, 1984c	
	Rodents	Single oral dose		Mortality	50	10 [a]	50		Eisler, 1987	
BBFANT	Rodents	Oral (chronic)	NS	Carcinogenicity		400 [b]	40		Eisler, 1987	
BKFANT	Rodents	Oral (chronic)	NS	Carcinogenicity		720 [b]	72		Eisler, 1987	
CTIRY	Rodents	Oral (chronic)	NS	Carcinogenicity		990 [b]	99		Eisler, 1987	
DBAIIA	Rodents	Oral (chronic)	NS	Carcinogenicity		0.06 [b]	0.006		Eisler, 1987	
DNBP	Rat	Oral (chronic)	1 year	Mortality		6000 [b]	600	125	IRIS, 1991	
DNOP	Rat	Oral (chronic)	7-12 months	Increased kidney weight		1750 [b]	175		USEPA, 1992	
DEP	Rat	Single oral dose		Mortality	8600	1720 [a]	172 [b]		NIOSH, 1985	
	Rat	Oral (chronic)	16 weeks	Growth/food ingestion/or gan weight changes			3160	750	IRIS, 1991	



**TABLE Q-4**  
**SUMMARY OF INGESTION TOXICITY DATA FOR TERRESTRIAL WILDLIFE**  
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CHEMICAL	TEST SPECIES	TEST TYPE	DURATION	EFFECT	ACUTE*		CHRONIC*		REFERENCE
					ORAL LD50 (mg/kg BW)	ACUTE ORAL RISK CRITERIA (mg/kg BW)	LOAEL (mg/kg BW/day)	NOAEL (mg/kg BW/day)	
24DNT (also surrogate for 26DNT)	Mouse	Single oral dose		Mortality	790	158 [a]		16 [b]	NIOSH, 1985
	Mouse	Oral (chronic)	24 months	Liver dysplasia				95	ATSDR, 1988a
	Rat	Single oral dose		Mortality	268	54 [a]			NIOSH, 1985
	Rat	Oral (chronic)	24 months	Anemia				40	ATSDR, 1988a
DPA	Guinea pig	Single oral dose		Mortality	1300				NIOSH, 1985
	Dog	Oral (subchronic)	13 weeks	Mortality	25	5 [a]		1 [b]	ATSDR, 1988a
	Dog	Oral (chronic)	24 months	Biliary hyperplasia				10	ATSDR, 1988a
	Rat	Oral (chronic)	2 year	Kidney lesions		310 [b]		31	IRIS, 1992
NNDMEA	Rat	Oral (chronic)	2 generation	Reduced litter size and weight of young		1250 [b]		125	IRIS, 1992
	Dog	Oral (chronic)	2 year	Low body weight gain, high liver/kidney weights		250 [b]		25	IRIS, 1992
	Rat	Single oral dose		Mortality	23	4.6 [a]		0.46 [b]	ATSDR, 1989
	Mink	Oral (chronic)	33 days	Hepatic necrosis		3.2 [b]		0.32	ATSDR, 1989
NNDPA	Dog	Oral (chronic)	2 years	Liver and kidney effects		25 [b]		2.5	USEPA, 1989
	Rat	Oral (chronic)	2 years	Bladder toxicity		500 [b]		50	ATSDR, 1988b
	Mouse	Oral (chronic)	2 years	Bladder toxicity		3000 [b]		300	ATSDR, 1988b
	Rat	Single oral dose		Mortality	480	96 [a]		9.6 [b]	ATSDR, 1989a
FANT	Rat	Oral (chronic)	30 weeks	Mortality		51 [b]		5.1	ATSDR, 1989a
	Rodents	Single oral dose		Mortality	2000	400 [a]		40 [b]	Eisler, 1987
	Mouse	Oral (chronic)	90 days	Liver weight/physiological changes				250	IRIS, 1990
	Mouse	Oral (chronic)	13 weeks	Hematological changes		2500 [b]		250	IRIS, 1990
FLRENE	Mouse	Oral (chronic)	NS	Carcinogenicity		720 [b]		72	Eisler, 1987
	Rodents	Oral (chronic)		Mortality	1630	330 [a]		33 [b]	NIOSH, 1985
	Rat	Single oral dose		Mortality	533	110 [a]			ATSDR, 1990
	Mouse	Single oral dose		Mortality				35.7	USEPA, 1990a
NAP (also surrogate for BGHIPPY)	Rat	Oral (chronic)	13 weeks	Decreased body weight gain				41	USEPA, 1989
	Rat	Oral (chronic)	100 weeks	Ocular lesions					Ellis et al., 1978
	Rat	Oral (chronic)		NOAEL		90000 [b]		9000 [d]	Ellis et al., 1978
	Rat	Oral (chronic)		NOAEL					Eisler, 1987
PIANTR	Rodents	Single oral dose		Mortality	700	140 [a]		14 [b]	NIOSH, 1985
	Mouse	Single oral dose		Mortality	800	140 [a]			IRIS, 1990
	Mouse	Oral (chronic)	13 weeks	Renal effects				125	NIOSH, 1985
	Rat	Single oral dose		Mortality	2700	540 [a]			IRIS, 1991
CCL <sub>4</sub>	Rat	Oral (chronic)	78 weeks	Mortality		4880 [b]		488	

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CHEMICAL	TEST SPECIES	TEST TYPE	DURATION	EFFECT	ACUTE*			CHRONIC*		NOAEL REFERENCE
					ORAL LD50 (mg/kg BW)	RISK CRITERIA (mg/kg BW)	LOAEL (mg/kg BW/day)	NOAEL (mg/kg BW/day)		
NG	Mouse	Oral (chronic)	24 months	NOAEL		315 [b]	315	115	Ellis et al., 1978a	
	Rat	Oral (chronic)	24 months	Hepatotoxicity					Ellis et al., 1978a	
	Dog	Oral (acute)	5 days	Methemoglobinemia		25	3 [b]		Ellis et al., 1978a	
	Dog	Oral (subchronic)	4 months	NOAEL				1	Ellis et al., 1978a	
INORGANIC COMPOUNDS										
AL	Mouse	Oral (chronic)	2-3 years	Reduced body weight gains of newborns					NIOSH, 1985	
	Rat	Oral (subchronic)	15 days	Reduced growth		1000 [b]	425		Bernuzzi et al., 1989	
	Rat	Dermal (acute)	60 min.	Mortality		48.4	100		ATSDR, 1990a	
	Rat, Rabbit, Cat	Oral (acute)		Mortality	1000	2245	20 [b]		ATSDR, 1990a	
	Rabbit	Oral (subchronic)	36 days	Renal damage		3180 [b]	224.5 [b]		ATSDR, 1990a	
	Dog	Oral (subchronic)	11 weeks	Bone deformity and softening			318		ATSDR, 1990a	
	Rat	Oral (chronic)	330 days	Bone loss, reduced body weight			936		ATSDR, 1990a	
SB	Rat	Oral (chronic)	NS	Longevity; blood glucose; cholesterol		4 [b]	0.35		IRIS, 1991	
AS	Rat	Oral (?)	NS	Weight loss		75 [b]	7.5		USEPA, 1984	
	Rat	Oral (chronic)		Mortality	323				Eisler, 1988	
	Mallard	Single oral dose		Mortality	47.6	9.5 [a]	1.0 [b]		Eisler, 1988	
	California quail	Single oral dose		Mortality	366				Eisler, 1988	
	Pheasant	Single oral dose		Mortality		2500 [b]	250 [d]		USEPA, 1984	
BA	Dog	Oral (chronic)	NS	Mortality					IRIS, 1990	
	Mouse	Oral (chronic)	lifetime	NOEL					IRIS, 1990	
	Rat	Oral (chronic)	16 months	NOEL					IRIS, 1990	
	Rat	Oral (chronic)	lifetime	NOEL		10 [b]	1 [d]		IRIS, 1990	
	Rat	Oral (chronic)	13 weeks	NOEL					IRIS, 1990	
BE	Rat	Single oral dose		Mortality	10	2 [a]			USEPA, 1985	
	Rat	Oral (chronic)	NS	Increase in lung sarcomas					USEPA, 1985	
BR	Rat	Oral (acute)		Mortality	3500	700 [a]	0.22		Sax, 1984	
CD	Mouse	Oral (chronic)	18 months	Histopathological effects		18 [b]	1.75		ATSDR, 1988c	
	Mouse	Oral (subchronic)	28 days	Alteration in blood chemistry					Eisler, 1985	
	Mouse (young)	Oral (chronic)	28 days	Blood chemistry altered					Eisler, 1985	
	Rat	Single oral dose		Mortality	250	50 [a]			Eisler, 1985	
	Rat	Single oral dose		Testicular damage					Eisler, 1985	
	Guinea pig	Single oral dose		Mortality	150	30 [a]	3 [b]		Eisler, 1985	
	Japanese quail	Oral (subchronic)	6 weeks	Bone marrow hypoplasia		76 [b]	7.6		Eisler, 1985	
	Mallard	Oral (chronic)	90 days	Egg production suppressed					Eisler, 1985	
	Mallard	Oral (chronic)	90 days	NOEL		100 [b]	10 [d]		Eisler, 1985	
	Mallard (young)	Oral (chronic)	12 weeks	Kidney lesions				20	Eisler, 1985	

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CHEMICAL	TEST SPECIES	TEST TYPE	DURATION	EFFECT	ACUTE*		CHRONIC*	
					ORAL LD50 (mg/kg BW)	ACUTE ORAL RISK CRITERIA (mg/kg BW)	LOAEL (mg/kg BW/day)	NOAEL REFERENCE (mg/kg BW/day)
CL	Rat	Oral (acute)		Mortality	3000	600 [a]	60 [b]	Sex, 1984
	Rabbit	Oral (acute)		Mortality	8000	1600 [a]	160 [b]	Sex, 1984
	Mouse	Oral (chronic)	13 weeks	Testicular degeneration		60 [b]	5.7	ATSDR, 1991
CR (Q+3)	Rat	Oral (chronic)	90 days	Hepatotoxicity			1400	USEPA, 1989
	Rat	Oral (chronic)	840 days	Various toxicological parameters			1468	IRIS, 1991
	Rabbit	Oral (chronic)	6 weeks	Liver and blood chemistry effects				Eisler, 1986
(Q+6)	Chicken	Oral (chronic)	32 days	Growth, survival		17 [b]	1.7	Eisler, 1986
	Black duck	Oral (chronic)	5 months	Growth patterns altered				8
	Japanese quail	Oral (acute)	5 days	Mortality	126 [c]	25 [a]	3.5	Eisler, 1986
(Potassium dichromate)	Rat	Single oral dose		TDLo for reproductive effects			2.5 [b]	Hill and Camardese, 1986
	Rat	Oral (chronic)	22 weeks	Fetotoxicity; CNS abnormalities		152	15.2 [b]	NIOSH, 1985
	Rat	Oral (chronic)	35 weeks	Pre-implantation mortality		12 [b]	1.21	NIOSH, 1985
CU	Swine	Oral (chronic)	9 months	Mortality			1.4	USEPA, 1980
	Mallard	Oral (acute)	29 days	No effect on survivorship		2.09	0.2 [b]	Demayo et al., 1983
	Mouse	Oral (chronic)	NS	Reduced success of implanted ova			1.5	Eisler, 1988a
PB	Rat	Single oral dose		Mortality	12	2 [a]		Eisler, 1988a
	Rat	Single oral dose		LDLO	17	3 [a]		Eisler, 1988a
	Rat	Oral (acute)	Days 12-14 (preg)	Increased fetal resorption rate; decreased fetal BW		2.5	0.3 [b]	McGinn and Becker, 1972
	Rat	Oral (acute)	Days 5-15 (preg)	Increased resorptions/dam		1	0.1 [b]	Kennedy et al., 1975
	Rat	Oral (subchronic)	3 weeks	Increased locomotor activity		1.5 [c]	0.2 [b]	Eisler, 1988a
	Rat	Oral (chronic)	2 years	Renal tumors			210 [c]	ATSDR, 1988d
	Rat	Oral (chronic)	NS	Increased locomotor activity			25	Eisler, 1988a
	Rabbit	Single oral dose		LDLO	24	5 [a]	0.5 [b]	ATSDR, 1988d
	Rabbit	Oral (chronic)	NS	Mortality		5.1 [b]	0.51 [c]	USEPA, 1988
	Chicken	Oral (subchronic)	4 weeks	Growth rate suppressed			169 [c]	Eisler, 1988a
	Ringed turtle - dove	Oral (acute)	NS	Some mortality; kidney damage		15 [a]	2.1 [b]	Eisler, 1988a
	Mallard	Single oral dose		Mortality	107	21 [a]	3.0 [b]	Eisler, 1988a
	Mallard	Oral (subchronic)	NS	Some mortality and ALAD decrease		30 [a]		Eisler, 1988a
	Mallard	Oral (chronic)	12 weeks	Decrease in ALAD activity			1.75 [c]	Eisler, 1988a
	Japanese quail	Single oral dose		Mortality	24.6	4.9 [a]		Eisler, 1988a
	Starling	Oral (acute)	11 days	Reduced food consumption			2.8	Eisler, 1988a
	Kestrel (nestlings)	Oral (acute)	10 days	Abnormal development		125	12.5 [b]	Eisler, 1988a
	Kestrel (nestlings)	Oral (acute)	10 days	ALAD depression		25	2.5 [b]	Eisler, 1988a
	Kestrel (nestlings)	Oral (acute)	10 days	Mortality and developmental effects		625	62.5 [b]	Eisler, 1988a
	Kestrel	Oral (chronic)	5 months	NOEL				0.89 [c] Eisler, 1988a

TABLE Q-4  
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CHEMICAL	TEST SPECIES	TEST TYPE	DURATION	EFFECT	ACUTE*		CHRONIC*	
					ORAL LD50 (mg/kg BW)	ACUTE ORAL RISK CRITERIA (mg/kg BW)	LOAEL (mg/kg BW/day)	NOAEL (mg/kg BW/day)
PB (continued)	Kestrel	Oral (chronic)	5 months	Blood ALAD reduced 80%		44 [b]	4.4 [c]	Eisler, 1988a
	Cattle (calves)	Oral (subchronic)	105 days	Mortality			6	Eisler, 1988a
	Horse	Oral (chronic)	NS	Mortality			2.4	Eisler, 1988a
	Dog	Oral (acute)	NS	LDLO		300	30 [b]	ATSDR, 1988d
	Dog	Oral (subchronic)	180 days	Anorexia and convulsions			3	Eisler, 1988a
	Mouse	Single oral dose		Mortality	22			NIOSH, 1985
	Mouse	Oral (subchronic)	18 days	Mortality; neurological symptoms			6.3	Suzuki, 1979
	Mouse	Oral (subchronic)	38 days	Mortality; neurological symptoms			5	Suzuki, 1979
	Mouse	Oral (subchronic)	50 days	Embryotoxicity and teratogenicity			0.9	Suzuki, 1979
	Mouse	Oral (subchronic)	45 days	Hypophagia, weight loss, weakness of hind legs			1	Suzuki, 1979
HG	Mouse	Oral (subchronic)	Day 6-17 (gest)	Stillbirths and neonatal death			4	Suzuki, 1979
	Mouse	Oral (subchronic)	Day 0-18 (gest)	Embryolethality and teratogenicity			0.7	Suzuki, 1979
	Rat	Oral (subchronic)	Day 6-14 (gest)	Retarded fetus growth and teratogenicity			4	Suzuki, 1979
	Rat	Oral (subchronic)	Gest. + 16 days	Behavioral changes in offspring			0.12 [c]	Suzuki, 1979
	Rat	Oral (chronic)	NS	Reduced fertility			0.5	Suzuki, 1979
	Rat	Oral (chronic)	38 days	Adverse behavioral change	18	3.6 [a]	0.16 [c]	Eisler, 1987a
	Rat	Single oral dose		Mortality			0.36 [b]	Eisler, 1987a
	Pig	Oral (chronic)	Pregnancy	High incidence of stillbirths			0.5	NIOSH, 1985
	House sparrow	Single oral dose		Mortality				Eisler, 1987a
	Rock dove	Single oral dose		Mortality	12.6	2.5 [a]		Eisler, 1987a
	Pigeon	Oral (subchronic)	17 days	Behavioral alterations	22.8	4.6 [a]	3	Eisler, 1987a
	Pigeon	Oral (subchronic)	5 weeks	Behavioral alterations			1	Eisler, 1987a
	Starling	Oral (chronic)	8 weeks	Kidney lesions			0.25 [c]	Eisler, 1987a
	Chicken	Single oral dose		Mortality	20	4 [a]		Eisler, 1987a
	Bantam chicken	Single oral dose		Mortality	190	38 [a]		Finreite, 1979
	Prairie chicken	Single oral dose		Mortality	11.5	2 [a]		Finreite, 1979
	Chukar	Single oral dose		Mortality	26.9	5 [a]		Eisler, 1987a
	Columbian	Single oral dose		Mortality	11	2 [a]		Eisler, 1987a
	Mallard	Single oral dose		Mortality	2.2	0.4 [a]		Eisler, 1987a
HG	Mallard	Oral (chronic)	3 Generations	Behavioral and reproductive deficiencies			0.007 [c]	Eisler, 1987a
	Black duck	Oral (chronic)	NS	Behavioral effects in offspring			0.036 [c]	Finreite, 1979
	Fulvous whistling du	Single oral dose	28 weeks	Reproduction inhibited, brain lesions			0.22 [c]	Eisler, 1987a
	Northern bobwhite	Single oral dose		Mortality	37.8	7.6 [a]		Eisler, 1987a
	Bobwhite quail	Single oral dose		Mortality	23.8	4.8 [a]		Eisler, 1987a
	Bobwhite quail	Oral (acute)	5 days	Mortality	523	105 [a]		Hill et al., 1975

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CHEMICAL	TEST SPECIES	TEST TYPE	DURATION	EFFECT	ACUTE*			CHRONIC*		
					ORAL LD50 (mg/kg BW)	ACUTE ORAL RISK CRITERIA (mg/kg BW)	LOAEL (mg/kg BW/day)	NOAEL (mg/kg BW/day)	REFERENCE	
HIG (continued)	Japanese quail	Single oral dose		Mortality	14.4	2.9 [a]			Eisler, 1987a	
	Japanese quail	Oral (su bchronic)	3 weeks	Depressed gonad weights			0.81 [c]		Eisler, 1987a	
	Japanese quail	Oral (su bchronic)	9 weeks	Alterations in brain and plasma enzyme activities			0.10 [c]		Eisler, 1987a	
	Japanese quail	Oral (chronic)	NS	Reproductive effects			5.0 [c]		Fimreite, 1979	
	Gray partridge	Single oral dose		Mortality	17.6	3.5 [a]			Eisler, 1987a	
	Gray pheasant	Oral (chronic)	30 days	Reduced reproductive ability			0.64		Eisler, 1987a	
	Ring-necked pheasant	Single oral dose		Mortality	11.5	2.3 [a]			Eisler, 1987a	
	Mule deer	Single oral dose		Mortality	17.9	3.6 [a]			Eisler, 1987a	
	Rhesus monkey	Oral (chronic)	Pregnancy	Maternally toxic and abortifacient	2	0.4 [a]		0.5	Eisler, 1987a	
	River otter	Single oral dose		Mortality	1	0.2 [a]			Eisler, 1987a	
	Mink	Single oral dose		Mortality			0.029 [c]		Eisler, 1987a	
	Mink	Oral (su bchronic)	2 months	Mortality			0.25		Eisler, 1987a	
	Cat	Oral (chronic)	Day 10-58 (gest)	Increased incidence of anomalous fetuses					Eisler, 1987a	
	Dog	Oral (chronic)	Pregnancy	High incidence of stillbirths					Eisler, 1987a	
NN	Mouse	Oral (su bchronic)	6 months	Mortality					2300 ATSDR, 1990b	
	Mouse	Oral (su bchronic)	90 days	Delayed growth of testes			140		ATSDR, 1990b	
	Mouse	Oral (chronic)	103 weeks	Mortality			4050 [d]		810 ATSDR, 1990b	
	Rat	Single oral dose		Mortality	410				ATSDR, 1990b	
	Rat	Oral (acute)	20 day	Mortality	225	45 [a]			ATSDR, 1990b	
	Rat	Oral (su bchronic)	10 weeks	Hepatic effects			4.5 [b]		ATSDR, 1990b	
	Rat	Oral (su bchronic)	20 days	Decreased litter weight during gestation		1240			12 ATSDR, 1990b	
	Rat	Oral (chronic)	103 weeks	Mortality			930		620 ATSDR, 1990b	
	Guinea pig	Single oral dose		Mortality	400				ATSDR, 1990b	
	Monkey	Oral (chronic)	18 months	Weakness, rigidity					USEPA, 1984e	
	Mouse	Oral (chronic)	2 years	Birth weight decrease; increase in abortions	67	13.4 [a]		25	ATSDR, 1990b	
	Rat	Single oral dose		Mortality					ATSDR, 1987	
	Rat	Oral (su bchronic)	91 days	Mortality					ATSDR, 1987	
	Rat	Oral (chronic)	2 years	Decreased body weight gain					ATSDR, 1987	
NTT	Japanese quail	Oral (acute)	5 days	NOEL	504 [c]	100.7 [a]			Hill and Camardese, 1986	
	Dog	Oral (chronic)	2 years	Histologic lesions in bone marrow		625 [b]			ATSDR, 1987	
	Mouse	Oral (su bchronic)	3 weeks	Elevated methemoglobin levels		1330 [b]			USEPA, 1985	
	Mouse	Oral (su bchronic)	3 weeks	NOAELS					USEPA, 1985	
	Rat	Oral (chronic)	6 months	Spleen hemorrhages		2500 [b]		250	USEPA, 1985	
	Rat	Oral (chronic)								

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CHEMICAL	TEST SPECIES	TEST TYPE	DURATION	EFFECT	ACUTE*		CHRONIC*	
					ORAL LD50 (mg/kg BW)	ACUTE ORAL RISK CRITERIA (mg/kg BW)	LOAEL (mg/kg BW/day)	NOAEL REFERENCE (mg/kg BW/day)
AO	Rat	Oral (chronic)	NS	Sclerosis		0.04 [c]	0.004 [b]	Eisler, 1985a
	Rat	Oral (chronic)	NS	Histological changes in heart and kidney			0.045	Eisler, 1985a
	Japanese quail	Oral (chronic)	NS	Reduced egg hatching		0.6 [c]	0.06 [b]	Eisler, 1985a
	Mallard	Oral (subchronic)	3 months	Reduced hatchability			1.75	Eisler, 1985a
	Horse	Single oral dose		MLD	3.3			Eisler, 1985a
SO4 (Mg, Na)	Mouse	Intraperitoneal (acute)		Mortality	34			NIOSH, 1985
	Rat	Oral (chronic)	2 week	Mortality		6.8 [a]		ATSDR, 1991
	Rat	Oral (chronic)	37 week	Weight gain		3624 [b]	362.4 [d]	ATSDR, 1991
	Mouse	Oral (chronic)	125 days	Increased hyperactivity		181 [b]	18.1	ATSDR, 1991
	Mouse	Single oral dose		Mortality		3000	300 [b]	NIOSH, 1985
SN	Rabbit	Single oral dose		Mortality		1198	120 [b]	NIOSH, 1985
	Rat	Single oral dose		Mortality	188	37.6 [a]	3.76 [b]	Eisler, 1989
	Rat	Oral (chronic)	13 weeks	NOEL				20 Eisler, 1989
	Rat	Oral (chronic)	12 days	Kidney damage			0.1	2 Eisler, 1989
	Rat	Oral (subchronic)	90 days	NOEL				Eisler, 1989
V	Mallard	Oral (subchronic)	NS	Vacuolization of spinal chord		35 [b]	3.5	Eisler, 1989
	Chicken	Oral (chronic)	15 weeks	Muscular weakness			12.9	Eisler, 1989
	Chinese quail	Oral (subchronic)	2 weeks	NOEL				15.1 Eisler, 1989
	Rat	Oral (chronic)	2.5 years	Decreased hair cystine			4 [d]	0.89 IRIS, 1989
	Rat	Oral (chronic)	103 days	Decreased hair cystine, hemoglobin		25 [b]	2.5	Hill and Camardese, 1986
ZN	Japanese quail	Oral (acute)	5 days	Mortality	69 [c]	10 [a]	1 [b]	SAX, 1984
	Rat	Single oral dose		Mortality	2510	500 [a]		Llobet et al., 1988
	Rat	Oral (subchronic)	NS	Kidney toxicity			140	

**TABLE Q-4**  
**SUMMARY OF INGESTION TOXICITY DATA FOR TERRESTRIAL WILDLIFE**  
**REMEDIAL INVESTIGATION**  
**BADGER ARMY AMMUNITION PLANT**

CHEMICAL	TEST SPECIES	TEST TYPE	DURATION	EFFECT	ACUTE*		CHRONIC*	
					ORAL LD50 (mg/kg BW)	RISK CRITERIA (mg/kg BW)	LOAEL (mg/kg BW/day)	NOAEL REFERENCE (mg/kg BW/day)

**NOTES:**

\* Shaded values are Reference Toxicity Values (RTV)

[a] For chemicals lacking LOAEL or NOAEL data, an Acute Oral Criterion (AOC) is calculated by applying a factor of 0.2 to the acute LD50; this value is expected to protect 99.9% of the exposed population from acute effects (USEPA, 1986).

[b] Estimated by applying an acute-chronic ratio of 10.

[c] Converted to dose normalized to body weight by multiplying by ingestion rate and dividing by body weight.

The following ingestion rate and body weight data were used:

Species	Ingestion Rate (kg/day)	Body Weight (kg)	Reference
Rat	0.015	0.25	NIOSH, 1985
Rabbit	0.059	2.2	USEPA, 1988
Chicken	0.106	1.16	USEPA, 1988
Bobwhite	0.015	0.17	Kenaga, 1973
California quail	0.014 [e]	0.139	USEPA, 1988
Mallard Duck	0.09	1.25	Ternes, 1987
Duck	0.112 [e]	1.6	USEPA, 1988
Starling	0.01	0.0437	USEPA, 1988
Kestrel	0.01	0.179	USEPA, 1988
Screech Owl	0.0086	0.169	USEPA, 1988
Mink	0.0465	1.613	USEPA, 1988
Mouse	0.0035	0.03	USEPA, 1988
Dog	0.5	14.47	USEPA, 1988

[d] Estimated by applying a LOAEL-NOAEL ratio of 5 (Newell, et al., 1987).

[e] Ingestion rate estimated from body weight using allometric equation for chickens in USEPA, 1988.

BW = Body Weight

LOAEL = Lowest Observed Adverse Effect Level

NOAEL = No Observed Adverse Effect Level

Table Q-5  
Compounds Detected  
Propellant Burning Ground Surface Soil (0-2')  
Units: ug/g

Remedial Investigation  
Badger Army Ammunition Plant

Compound	Frequency	Maximum	Minimum	Retained for Risk Assessment		Exposure Point Concentration **
				(Y/N)?	Reason *	
24DNT	16 : 114	53.3	2.77	Y		10.7
26DNT	2 : 114	4.25	3.41	N	4	
2MNAP	2 : 13	0.452	0.122	Y		0.452
ACET	1 : 58	0.006	-	N	4	
AG	3 : 108	25.8	2.02	N	4	
AS	83 : 108	64	2.88	Y		9.45
B2EHP	1 : 13	6.2	-	Y		6.2
BAANTR	1 : 13	0.204	-	Y		0.204
BE	81 : 108	2.29	0.494	N	1	
C6H6	8 : 114	2.64	0.199	Y		0.42
CCL3F	4 : 114	0.005	0.003	N	4	
CD	3 : 108	4.48	1.7	N	4	
CHRY	1 : 13	3.68	-	Y		3.68
CR	108 : 108	89.8	7.15	Y		49.8
CU	108 : 108	2700	9.57	Y		344
DEP	7 : 13	6.2	0.568	Y		6.2
DNBP	4 : 13	6.35	2.06	Y		6.35
FANT	2 : 13	0.2	0.145	Y		0.2
HG	31 : 108	7.7	0.058	Y		0.334
MEK	7 : 64	0.01	0.006	N	2	
NI	108 : 108	63.9	6.57	Y		27.3
NNDPA	3 : 13	30.8	1.22	Y		30.8
PB	108 : 108	3300	12	Y		2700
PHANTR	3 : 13	1.32	0.11	Y		1.32
PYR	1 : 13	0.168	-	Y		0.168
SB	1 : 108	404	-	N	4	
SE	10 : 108	2.03	0.581	Y		0.618
TL	2 : 108	2.28	1.19	N	1, 4	
ZN	108 : 108	5200	27	Y		1040

Footnotes:

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential nutrient or without known toxicity.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum

Note: Assessment of surface soil contamination was performed using samples PBS-91-01 through PBS-91-108. In addition, the upper portions of samples PBS-91-109 through PBS-91-114 were used to assess contamination of surface soil by 24DNT, 26DNT, C6H6, and CCL3F.



**Table Q-6**  
**Compounds Detected**  
**Final Creek**  
**Settling Ponds and Spoils Disposal Area Surface Soil (0-2')**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	<u>Concentration **</u>
AL	8 : 8	14000	890	N	1	
PB	8 : 8	40	3.6	Y		40
K	8 : 8	920	26	N	1,3	
NA	8 : 8	180	18	N	1,3	
SN	7 : 8	63	25	Y		63
NIT	8 : 8	11	1.6	Y		11
NH3	8 : 8	1800	24	Y		1800
SO4	4 : 8	260	28	Y		260
24DNT	5 : 8	6	0.17	Y		6
26DNT	6 : 8	40	1.6	Y		40
DEP	2 : 8	0.13	0.11	Y		0.13
DNBP	5 : 8	26	1.7	Y		26
DPA	6 : 8	15	0.22	Y		15
2NNDPA	3 : 8	2	0.57	Y		2
NC	3 : 8	740	100	Y		740

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant.  
                   \* 3 = essential nutrient or without known toxicity.  
                   \* 4 = frequency of detection less than 5 %.  
                   \*\* 95th percentile or maximum.

Note:           Assessment of surface soil contamination (0 to 2 feet) was performed  
                   using data from samples FC-1 through FC-8.

**Table Q-7**  
**Compounds Detected**  
**Settling Pond 1**  
**Settling Ponds and Spoils Disposal Area Surface Soil (0-2')**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u> <u>(Y/N)?</u>	<u>Reason *</u>	<u>Exposure Point</u> <u>Concentration **</u>
AL	17 : 17	27000	1400	N	1	
PB	16 : 17	180	5.1	Y		180
K	14 : 14	1100	69	N	1, 3	
NA	14 : 14	150	17	N	1, 3	
SN	17 : 17	57	0.45	Y		57
NIT	14 : 16	13	0.2	Y		13
NH3	14 : 14	740	53	Y		740
SO4	8 : 18	2500	58	Y		2500
24DNT	5 : 15	172	0.03	Y		172
26DNT	6 : 14	26	0.16	Y		26
DEP	1 : 15	460		Y		460
DNBP	6 : 15	14	0.1	Y		14
DPA	6 : 14	10	0.24	Y		10
2NNDPA	3 : 14	0.97	0.72	Y		0.97
NC	7 : 15	60000	180	Y		60000

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant.  
                   \* 3 = essential nutrient or without toxicity.  
                   \* 4 = frequency of detection less than 5 %.  
                   \*\* 95th percentile or maximum.

Note:           Assessment of surface soil contamination (0 to 2 feet) was performed  
                   using data from samples FPI-1 through FPI-14, and S1201 through S1204.

**Table Q-8**  
**Compounds Detected**  
**Settling Pond 2**  
**Settling Ponds and Spoils Disposal Area Surface Soil (0-2')**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
AL	3 : 3	40000	12000	N	1	250
PB	3 : 3	250	95	Y		
K	3 : 3	600	370	N	1, 3	
NA	3 : 3	120	72	N	1, 3	
SN	3 : 3	53	22	Y		53
NIT	3 : 3	43	14	Y		43
NH3	3 : 3	840	260	Y		840
SO4	1 : 3	64		Y		64
24DNT	1 : 4	7.6		Y		7.6
DEP	1 : 4	135		Y		135
DNBP	1 : 4	0.74		Y		0.74
DPA	1 : 3	1.5		Y		1.5
NC	2 : 3	280	260	Y		280

Footnotes:

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential nutrient or without known toxicity.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum.

Note: Assessment of surface soil contamination (0 to 2 feet) was performed using data from samples FPII-1 through FPII-3 and S1205.

Table Q-9  
Compounds Detected  
Settling Pond 3  
Settling Ponds and Spoils Disposal Area Surface Soil (0-2")  
Units: ug/g

Remedial Investigation  
Badger Army Ammunition Plant

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
AL	15 : 15	34000	2900	N	1	
PB	15 : 15	34	6.7	N	1	
K	15 : 15	1300	140	N	1,3	
NA	15 : 15	160	1.1	N	1,3	
SN	15 : 15	72	23	Y		72
NIT	15 : 15	4.9	0.39	Y		4.9
NH3	15 : 15	520	21	Y		520
SO4	2 : 15	36	30	Y		36
24DNT	1 : 16	2.6		Y		2.6
26DNT	1 : 15	1.5		Y		1.5
DEP	1 : 16	44		Y		44
DNBP	5 : 16	17.4	2.5	Y		17.4
DPA	4 : 15	2.8	0.24	Y		2.8
NC	2 : 15	190	50	Y		190

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant.  
                   \* 3 = essential nutrient or without known toxicity.  
                   \* 4 = frequency of detection less than 5 %.  
                   \*\* 95th percentile or maximum

Note:           Assessment of surface soil contamination (0 to 2 feet) was performed  
                   using data from samples FPIII-1 through FPIII-15 and S1206.

Table Q-10  
Compounds Detected  
Settling Pond 4  
Settling Ponds and Spoils Disposal Area Surface Soil (0-2')  
Units: ug/g

Remedial Investigation  
Badger Army Ammunition Plant

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
AL	11 : 11	60000	1300	Y		60000
PB	11 : 11	300	8.4	Y		300
K	10 : 10	1900	25	N	1, 3	
NA	9 : 10	400	44	N	1, 3	
SN	11 : 11	77	1.1	Y		77
NIT	10 : 11	10	0.67	Y		10
NH3	10 : 10	960	29	Y		960
SO4	3 : 11	400	170	Y		400
DPA	1 : 10	0.36		Y		0.36
NC	2 : 11	1038	50	Y		1038

Footnotes:

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential nutrient or without known toxicity.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum

Note: Assessment of surface soil contamination (0 to 2 feet) was performed using samples FPIV-1 through FPIV-10 and S1207.

Table Q-11  
Compounds Detected  
Spoils Disposal Site 1  
Settling Ponds and Spoils Disposal Area Surface Soil (0-2')  
Units: ug/g

Remedial Investigation  
Badger Army Ammunition Plant

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
AL	5 : 5	44258	12487	N	1	
FE	5 : 5	35401	4162	N	1, 3	
PB	5 : 5	349	42	Y		349
K	5 : 5	1660	55	N	1, 3	
NA	5 : 5	199	90	N	1, 3	
SN	5 : 5	3.68	2.54	Y		3.68
ZN	5 : 5	212	63	Y		212
BR	2 : 2	12		Y		12
CL	5 : 5	19	13	Y		19
NIT	5 : 5	16	8	Y		16
SO4	5 : 5	146	33	Y		146
CH2CL2	3 : 3	0.01	0.034	Y		0.01
24DNT	3 : 3	12	0.51	Y		12
26DNT	1 : 1	1		Y		1
B2EHP	1 : 1	0.35		Y		0.35
DNBP	5 : 5	51	0.82	Y		51
DNOP	1 : 1	8.6		Y		8.6
DPA	4 : 4	24	0.34	Y		24
NC	5 : 5	11000	6000	Y		11000
NG	1 : 1	19		Y		19

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant.  
                   \* 3 = essential nutrient or without known toxicity.  
                   \* 4 = frequency of detection less than 5 %.  
                   \*\* 95th percentile or maximum.

Note:           Assessment of surface soil contamination (0 to 2 feet) was performed  
                   using samples SD1-1 through SD1-5.

Table Q-12  
Compounds Detected  
Spoils Disposal Site 2  
Settling Ponds and Spoils Disposal Area Surface Soil (0-2')  
Units: ug/g

Remedial Investigation  
Badger Army Ammunition Plant

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	Retained for Risk Assessment		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
AL	5 : 5	49398	4547	N	1	
FE	5 : 5	18674	15534	N	1, 3	
PB	5 : 5	373	239	Y		373
K	5 : 5	566	437	N	1, 3	
NA	5 : 5	235	123	N	1, 3	
SN	5 : 5	4.04	1.04	Y		4.04
ZN	5 : 5	748	148	Y		748
BR	1 : 1	4		Y		4
CL	5 : 5	23	16	Y		23
NIT	5 : 5	10	8	Y		10
SO4	5 : 5	130	80	Y		130
CH2CL2	3 : 3	0.012	0.024	Y		0.012
24DNT	4 : 4	1.3	0.48	Y		1.3
DNBP	5 : 5	5.8	0.98	Y		5.8
DPA	5 : 5	3.2	0.24	Y		3.2
NC	5 : 5	8000	5800	Y		8000

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant.  
                   \* 3 = essential nutrient or without known toxicity.  
                   \* 4 = frequency of detection less than 5 %.  
                   \*\* 95th percentile or maximum.

Note:           Assessment of surface soil contamination (0 to 2 feet) was performed  
                   using samples SD2-1 through SD2-5.

**Table Q-13**  
**Compounds Detected**  
**Spoils Disposal Site 3**  
**Settling Ponds and Spoils Disposal Area Surface Soil (0-2')**  
**Units ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
AL	10: 10	26530	7123	N	1	
FE	10: 10	15696	5224	N	1, 3	
PB	10: 10	67	24	Y		67
K	10: 10	1327	121	N	1, 3	
NA	10: 10	286	95	N	1, 3	
SN	10: 10	5.8	1.16	Y		5.8
ZN	10: 10	251	84	Y		251
CL	10: 10	17	10	Y		17
NIT	10: 10	22	9	Y		22
SO4	10: 10	75	29	Y		75
CH2CL2	1: 1	0.025		Y		0.025
24DNT	5: 5	1.1	0.24	Y		1.1
DNBP	9: 9	4	0.26	Y		4
DPA	5: 5	2.2	0.25	Y		2.2
NC	10: 10	3800	450	Y		3800

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant  
                   \* 3 = essential nutrient or without known toxicity.  
                   \* 4 = frequency of detection less than 5 %.  
                   \*\* 95th percentile or maximum

Note:           Assessment of surface soil contamination (0 to 2 feet) was performed  
                   using samples from SD3-1 through SD3-10.



**Table Q-14**  
**Compounds Detected**  
**Spoils Disposal Site 4**  
**Settling Ponds and Spoils Disposal Area Surface Soil (0-2")**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	<u>Concentration **</u>
AL	10 : 10	20865	11511	N	1	
FE	10 : 10	19894	13512	N	1, 3	
PB	10 : 10	120	22	Y		120
K	10 : 10	1819	415	N	1, 3	
NA	10 : 10	255	98	N	1, 3	
SN	10 : 10	1.64	0.63	Y		1.64
ZN	10 : 10	204	89	Y		204
CL	9 : 9	13	10	Y		13
NIT	10 : 10	12	4	Y		12
SO4	10 : 10	139	22	Y		139
CH2CL2	4 : 4	0.01	0.038	Y		.01
24DNT	1 : 1	0.7		Y		.7
B2EHP	1 : 1	0.32		Y		.32
DNBP	4 : 4	4.4	0.32	Y		4.4
DNOP	3 : 3	0.63	0.22	Y		0.63
DPA	1 : 1	1.1		Y		1.1
NC	9 : 9	3000	33	Y		3000

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant.  
                   \* 3 = essential nutrient or without known toxicity.  
                   \* 4 = frequency of detection less than 5 %.  
                   \*\* 95th percentile or maximum

Note:           Assessment of surface soil contamination (0 to 2 feet) was performed  
                   using samples from SD4-1 through SD4-10.

**Table Q-15**  
**Compounds Detected**  
**Spoils Disposal Site 5**  
**Settling Ponds and Spoils Disposal Area Surface Soil (0-2')**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason</u>	
AL	9 : 9	19436	3684	N	1	
FE	9 : 9	18922	10591	N	1, 3	
PB	8 : 8	102	23	Y		102
K	9 : 9	1336	111	N	1, 3	
NA	10 : 10	216	64	N	1, 3	
SN	10 : 10	1.94	0.63	Y		1.94
ZN	9 : 9	306	101	Y		306
BR	1 : 1	16		Y		16
CL	9 : 9	18	10	Y		18
NIT	10 : 10	18	7	Y		18
SO4	10 : 10	38	23	Y		38
CH2CL2	3 : 3	0.01	0.026	Y		.01
DNBP	7 : 7	6.5	0.33	Y		6.5
DNOP	1 : 1	0.2		Y		.2
DPA	3 : 3	2.4	0.22	Y		2.4
NC	8 : 8	11000	250	Y		11000

Footnotes:   \* 1 = within background range.  
                  \* 2 = laboratory or sampling contaminant.  
                  \* 3 = essential nutrient or without known toxicity.  
                  \* 4 = frequency of detection less than 5 %.  
                  \*\* 95th percentile or maximum

Note:           Assessment of surface soil contamination (0 to 2 feet) was performed  
                   using samples from SD5-1 through SD5-10.

Table Q-16  
Compounds Detected  
Rocket Paste Area Surface Soil (0-2')  
Units: ug/g

Remedial Investigation  
Badger Army Ammunition Plant

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
123PDA	1 : 72	19	-	N	4	
24DNT	12 : 72	810	3.15	Y		810
26DNT	10 : 72	32.5	0.783	Y		32.5
B2EHP	2 : 72	1.61	1.56	N	4	
BAANTR	4 : 72	0.666	0.173	Y		0.666
BBFANT	2 : 72	2.13	2.03	N	4	
BGHIPY	1 : 72	1.91	-	N	4	
CHRY	8 : 72	1	0.08	Y		1
CR	66 : 66	109	3.41	Y		109
DEP	37 : 72	49.8	0.652	Y		49.8
FANT	20 : 72	1.12	0.046	Y		1.12
HG	17 : 66	0.716	0.054	Y		0.716
NG	42 : 66	1500	0.709	Y		1500
NIT	65 : 66	120	1.36	Y		120
NNDMEA	7 : 72	0.302	0.022	Y		0.302
NNDNPA	5 : 72	0.23	0.096	Y		0.23
NNDPA	58 : 72	10000	0.092	Y		10000
PB	66 : 66	3500	8.5	Y		3500
PHANTR	14 : 72	0.279	0.076	Y		0.279
PYR	8 : 72	0.932	0.179	Y		0.932
SO4	17 : 66	22.9	6.21	Y		22.9

Footnotes:

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential nutrient or without known toxicity.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum

Note: Assessment of surface soil contamination (0 to 2 feet) was performed using samples from RPS-91-03 through RPS-91-68.

Table Q-17  
Compounds Detected  
Rocket Paste Area Pond Surface Water  
Units: ug/L

Remedial Investigation  
Badger Army Ammunition Plant

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
AL	2: 2	31400	5410	Y		31400
AS	2: 2	15	8.6	Y		15
BA	2: 2	290	121	Y		290
BE	1: 2	2.17	-	Y		2.17
CA	2: 2	38200	30800	N	3	
CL	2: 2	2730	2700	Y		2730
CR	1: 2	59.5	-	Y		59.5
CU	2: 2	79.1	21.3	Y		79.1
FE	2: 2	31700	7980	Y		31700
K	2: 2	44000	43000	N	3	
MG	2: 2	20900	14900	N	3	
MN	2: 2	503	152	Y		503
NA	2: 2	2000	1190	N	3	
NH3N2	2: 2	63.4	33.8	Y		63.4
NI	1: 2	40.7	-	Y		40.7
NIT	1: 2	10.5	-	Y		10.5
PB	2: 2	3100	910	Y		3100
SO4	2: 2	35000	32000	Y		35000
V	2: 2	57.1	22.3	Y		57.1
ZN	2: 2	151	34.9	Y		151

Footnotes:

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential nutrient or without known toxicity.
- \* 4 = frequency of detection less than 5 %
- \*\* 95th percentile or maximum

Note: Assessment of surface water contamination was performed using samples RPW-91-01 and RPW-91-02.

Table Q-18  
Compounds Detected  
Rocket Paste Pond Area Sediment  
Units: ug/g

Remedial Investigation  
Badger Army Ammunition Plant

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	Retained for Risk Assessment		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
CR	2: 2	45.7	33.8	Y		45.7
DEP	1: 2	2.46	-	Y		2.46
NG	1: 2	1.76	-	Y		1.76
NIT	2: 2	2.22	1.96	Y		2.22
NNDPA	2: 2	4.98	0.738	Y		4.98
PB	2: 2	2600	1100	Y		2600
SO4	2: 2	210	150	Y		210

Footnotes:

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential nutrient or without known toxicity.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum

Note: Assessment of sediment contamination was performed using samples from RPS-91-01 and RPS-91-02.

Table Q-19  
Compounds Detected  
Nitroglycerine Pond Surface Soil  
Units:ug/g

Remedial Investigation  
Badger Army Ammunition Plant

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
CR	2:2	39.5	32.2	N	1	
HG	1:2	2.4	-	Y		2.4
NG	2:2	15.8	9.39	Y		15.8
NH3	2:2	17.7	4.47	Y		17.7
PB	2:2	10000	2000	Y		10000

Footnotes:

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential nutrient or without known toxicity.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum

Note: Assessment of surface soil contamination was performed using samples from NPS-91-09 and NPS-91-10.

**Table Q-20**  
**Compounds Detected**  
**Nitroglycerine Pond Surface Water**  
**Units: ug/L**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
AL	2 : 2	3020	2140	Y		3020
AS	2 : 2	5.43	4.98	Y		5.43
BA	2 : 2	63.1	47.3	Y		63.1
CA	2 : 2	15200	11700	N	3	
CL	2 : 2	1930	1680	Y		1930
FE	2 : 2	3970	2920	Y		3970
HG	2 : 2	0.325	0.324	Y		0.325
K	2 : 2	15000	12800	N	3	
MG	2 : 2	5880	5340	N	3	
MN	2 : 2	207	81.7	Y		207
NA	2 : 2	8320	7790	N	3	
NH3N2	2 : 2	147	63.4	Y		147
PB	2 : 2	45.9	41.2	Y		45.9
SO4	2 : 2	4470	4070	Y		4470
V	2 : 2	8.37	6.62	Y		8.37

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant.  
                   \* 3 = essential nutrient or without known toxicity.  
                   \* 4 = frequency of detection less than 5 %.  
                   \*\* 95th percentile or maximum

Note:           Assessment of surface water contamination was performed using samples  
 NPW-91-01 and NPW-91-02.

Table Q-21  
Compounds Detected  
Nitroglycerine Pond Sediment  
Units: ug/g

Remedial Investigation  
Badger Army Ammunition Plant

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
CR	8: 8	40.5	4.9	Y		40.5
HG	8: 8	20	0.159	Y		20
NH3	8: 8	72.5	2.28	Y		72.5
PB	8: 8	410	32	Y		410

Footnotes:

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential nutrient or without known toxicity.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum

Note: Assessment of sediment contamination was performed using samples from NPS-91-01 through NPS-91-08.



**Table Q-22**  
**Compounds Detected**  
**Oleum Plant Surface Soil (0-2')**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
CR	1: 9	14.4	-	N	1	
FE	1: 9	16	-	N	1	
PB	1: 9	6.82	-	N	1	
NIT	3: 3	3.46	1.68	Y		3.46
SO4	3: 9	8500	1000	Y		8500

**Footnotes:**

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential nutrient or without known toxicity.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum

**Note:** Assessment of surface soil contamination (0 to 2 feet) was performed using samples from borings OPB-91-01 and OPB-91-06 through OPB-91-13.

**Table Q-23**  
**Compounds Detected**  
**Oleum Pond Sediment**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	<u>Concentration **</u>
CA	4 : 4	36900	4380	N	3	
NA	3 : 4	120	67.2	N	3	
NIT	4 : 4	50	14	Y		50
SO4	4 : 4	590	160	Y		590

Footnotes:   \* 1 = within background range.  
                   \* 2 = laboratory or sampling contaminant.  
                   \* 3 = essential nutrient or without known toxicity.  
                   \* 4 = frequency of detection less than 5 %.  
                   \*\* 95th percentile or maximum

Note:           Assessment of sediment contamination was performed using samples  
                   OPS-91-01 through OPS-91-04.

**Table Q-24**  
**Compounds Detected**  
**Ballistics Pond Surface Water**  
**Units: ug/L**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
AL	2 : 2	180	123	Y		180
BA	2 : 2	36.7	34.6	Y		36.7
CA	2 : 2	6510	6260	N	3	
CL	5 : 5	4050	2934.44	Y		4050
FE	2 : 2	315	217	Y		315
K	2 : 2	1940	1490	N	3	
MG	2 : 2	2920	2810	N	3	
MN	2 : 2	79.1	76.8	Y		79.1
NA	2 : 2	3780	3580	N	3	
NIT	3 : 5	51.4	11.223	Y		51.4
SO4	5 : 5	15000	8516.35	Y		15000
V	1 : 2	5.23	-	Y		5.23
ZN	2 : 2	67.9	35.4	Y		67.9

Footnotes:

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential for nutrient or without known toxicity.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum

Note: Assessment of surface water contamination was performed using samples BPW-91-01 and BPW-91-02.

**Table Q-25**  
**Compounds Detected**  
**Ballistics Pond Sediment**  
**Units: ug/g**

**Remedial Investigation**  
**Badger Army Ammunition Plant**

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	<u>Concentration **</u>
AL	6 : 6	58000	10200	Y		58000
B2EHP	2 : 6	6.1	1.27	Y		6.1
NH3	5 : 6	215	13.9	Y		215
NIT	1 : 6	5.16	-	Y		5.16
PB	6 : 6	54	2.07	Y		54
PHANTR	1 : 6	0.428	-	Y		0.428
SO4	6 : 6	490	62.7	Y		490

Footnotes:

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential nutrient or without known toxicity.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum

Note: Assessment of sediment contamination was performed using samples BPS-91-01 through BPS-91-06.

Table Q-26  
Compounds Detected  
Old Acid Area Surface Soil (0-2')  
Units: ug/g

Remedial Investigation  
Badger Army Ammunition Plant

<u>Compound</u>	<u>Frequency</u>	<u>Maximum</u>	<u>Minimum</u>	<u>Retained for Risk Assessment</u>		<u>Exposure Point Concentration **</u>
				<u>(Y/N)?</u>	<u>Reason *</u>	
CR	3 : 3	20.5	14.4	N	1	
MEK	2 : 3	0.006	-	N	2	
NI	3 : 3	56.9	17.3	Y		56.9
NIT	13 : 23	5.61	1.09	Y		1.79
PB	3 : 3	1500	4.87	Y		1500
SO4	16 : 23	20000	5.78	Y		18000

Footnotes:

- \* 1 = within background range.
- \* 2 = laboratory or sampling contaminant.
- \* 3 = essential nutrient or without known toxicity.
- \* 4 = frequency of detection less than 5 %.
- \*\* 95th percentile or maximum

Note: Assessment of surface soil contamination (0 to 2 feet) was performed using samples from borings OAB-91-01 through OAB-91-13.

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**APPENDIX R**  
**ECOLOGICAL RISK CALCULATIONS**

# ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

**PROPELLANT BURNING GROUND, BADGER ARMY AMMUNITION PLANT**

CHEMICAL	CONCENTRATION (mg/kg)
24DNT	1.1E+01
2MNAP	4.5E-01
AS	9.5E+00
BAANTR	2.0E-01
B2EHP	6.2E+00
C6H6	4.2E-01
CHRY	3.7E+00
CR	5.0E+01
CU	3.4E+02
DEP	6.2E+00
DNBP	6.4E+00
FANT	2.0E-01
HG	3.3E-01
NI	2.7E+01
NNDPA	3.1E+01
PB	2.7E+03
PHANTR	1.3E+00
PYR	1.7E-01
SE	6.2E-01
ZN	1.0E+03

Invert	Tissue		Plant	Tissue	
	BAF [a]	Level (mg/kg)		BAF [a]	Level (mg/kg)
1.0E+00	1.1E+01	1.0E+00	1.1E+01	1.1E+01	
1.0E+00	4.5E-01	1.0E-03	1.0E-03	4.5E-04	
1.0E+00	9.5E+00	2.0E-01	2.0E-01	1.9E+00	
1.0E+00	2.0E-01	2.2E-02	2.2E-02	4.5E-03	
1.0E+00	6.2E+00	4.3E-02	4.3E-02	2.7E-01	
1.0E+00	4.2E-01	1.0E+00	1.0E+00	4.2E-01	
1.0E+00	3.7E+00	2.2E-02	2.2E-02	8.1E-02	
1.6E-01	8.0E+00	1.0E-01	1.0E-01	5.0E+00	
9.3E+00	3.2E+03	1.0E+01	1.0E+01	3.4E+03	
1.0E+00	6.2E+00	5.3E-01	5.3E-01	3.3E+00	
1.0E+00	6.4E+00	6.5E-02	6.5E-02	4.1E-01	
1.0E+00	2.0E-01	5.7E-02	5.7E-02	1.1E-02	
3.4E-01	1.1E-01	1.0E+00	1.0E+00	3.3E-01	
1.9E+00	5.2E+01	3.2E+00	3.2E+00	8.7E+01	
1.0E+00	3.1E+01	6.0E-01	6.0E-01	1.8E+01	
2.4E+00	6.5E+03	2.0E-01	2.0E-01	5.4E+02	
1.0E+00	1.3E+00	1.0E-01	1.0E-01	1.3E-01	
1.0E+00	1.7E-01	5.9E-02	5.9E-02	9.9E-03	
1.0E+00	6.2E-01	1.0E+00	1.0E+00	6.2E-01	
7.3E+00	7.6E+03	1.0E+01	1.0E+01	1.0E+04	

Small Mammal	Small Bird	Herpetile
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
3.7E-01	5.6E-01	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
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1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
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1.0E+00	1.0E+00	1.0E+00
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1.0E+00	1.0E+00	1.0E+00
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1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
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1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
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1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E	

06-Nov-92

Table R-1  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
PROPELLANT BURNING GROUND, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]

CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
24DNT	1.9E+01	1.2E+00	9.1E-01	5.0E-01	1.6E+00
2MNAP	7.2E-01	3.9E-02	3.8E-02	1.8E-02	5.9E-02
AS	1.5E+01	8.7E-01	7.6E-01	2.6E-01	7.0E-01
BAANTR	3.2E-01	1.8E-02	1.7E-02	8.1E-03	2.7E-02
B2EHP	9.9E+00	5.5E-01	5.2E-01	2.5E-01	8.2E-01
C6H6	7.4E-01	4.6E-02	3.6E-02	2.0E-02	6.4E-02
CHRY	5.8E+00	3.2E-01	3.1E-01	1.5E-01	4.8E-01
CR	1.7E+01	1.0E+00	8.7E-01	5.1E-01	1.8E+00
CU	5.4E+03	3.4E+02	2.6E+02	1.4E+02	4.6E+02
DEP	1.0E+01	6.1E-01	5.2E-01	2.7E-01	8.9E-01
DNBP	1.0E+01	5.6E-01	5.3E-01	2.5E-01	8.4E-01
FANT	3.2E-01	1.8E-02	1.7E-02	8.0E-03	2.6E-02
HG	2.6E-01	1.8E-02	1.4E-02	3.1E-02	1.1E-01
NI	9.5E+01	6.3E+00	4.1E+00	1.7E+00	4.3E+00
NNDPA	5.2E+01	3.1E+00	2.6E+00	1.4E+00	4.4E+00
PB	1.0E+04	5.6E+02	5.0E+02	1.7E+02	4.3E+02
PHANTR	2.1E+00	1.2E-01	1.1E-01	5.3E-02	1.8E-01
PYR	2.7E-01	1.5E-02	1.4E-02	6.7E-03	2.2E-02
SE	1.1E+00	6.7E-02	5.3E-02	2.9E-02	9.5E-02
ZN	1.3E+04	8.5E+02	1.1E+03	2.1E+03	7.7E+03

Table R-1

## ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## PROPELLANT BURNING GROUND, BADGER ARMY AMMUNITION PLANT

## EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet					Home Range (acres)	Soil	Birds	Herpeto-fauna	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Herpeto-fauna	Birds							
<i>Short-tailed shrew</i> (Small Mammal)	85%	10%	0%	0%	0%	1.3	5%	0%	0%	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i> (Small Bird)	75%	20%	0%	0%	0%	5	5%	0%	0%	1.0E+00	0.0095	0.087
<i>Garter snake</i> (Herptile)	85%	0%	5%	0%	5%	5	5%	5%	0%	1.0E+00	0.023	0.27
<i>Red fox</i> (Pred. Mammal)	20%	10%	40%	15%	10%	250	5%	10%	15%	1.9E-01	0.23	4.9
<i>Red-tailed hawk</i> (Pred. Bird)	5%	5%	55%	10%	20%	500	5%	20%	10%	9.6E-02	0.23	1.5

## NOTES:

[a] Bioaccumulation data presented in: Appendix Q, Table Q-1

[b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.

[c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2

[d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)





TABLE R-2

## ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## PROPELLANT BURNING GROUND, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]

CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
24DNT	1.9E+01	1.2E+00	9.1E-01	9.6E-02	1.6E-01
2MNAP	7.2E-01	3.9E-02	3.8E-02	3.4E-03	5.7E-03
AS	1.5E+01	8.7E-01	7.6E-01	5.1E-02	6.8E-02
BAANTR	3.2E-01	1.8E-02	1.7E-02	1.5E-03	2.6E-03
B2EHP	9.9E+00	5.5E-01	5.2E-01	4.7E-02	7.8E-02
C6H6	7.4E-01	4.6E-02	3.6E-02	3.8E-03	6.2E-03
CHRY	5.8E+00	3.2E-01	3.1E-01	2.8E-02	4.6E-02
CR	1.7E+01	1.0E+00	8.7E-01	9.9E-02	1.7E-01
CU	5.4E+03	3.4E+02	2.6E+02	2.7E+01	4.4E+01
DEP	1.0E+01	6.1E-01	5.2E-01	5.2E-02	8.5E-02
DNBP	1.0E+01	5.6E-01	5.3E-01	4.9E-02	8.1E-02
FANT	3.2E-01	1.8E-02	1.7E-02	1.5E-03	2.5E-03
HG	2.6E-01	1.8E-02	1.4E-02	5.9E-03	1.0E-02
NI	9.5E+01	6.3E+00	4.1E+00	3.3E-01	4.2E-01
NNDPA	5.2E+01	3.1E+00	2.6E+00	2.6E-01	4.3E-01
PB	1.0E+04	5.6E+02	5.0E+02	3.2E+01	4.1E+01
PHANTR	2.1E+00	1.2E-01	1.1E-01	1.0E-02	1.7E-02
PYR	2.7E-01	1.5E-02	1.4E-02	1.3E-03	2.1E-03
SE	1.1E+00	6.7E-02	5.3E-02	5.6E-03	9.1E-03
ZN	1.3E+04	8.5E+02	1.1E+03	4.0E+02	7.4E+02

TABLE R-2  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
PROPELLANT BURNING GROUND, BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet					Home Range (acres)	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Herpeto-fauna	Birds				
<i>Short-tailed shrew</i> (Small Mammal)	85%	10%	0%	0%	0%	1.3	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i> (Small Bird)	75%	20%	0%	0%	0%	5	1.0E+00	0.0095	0.087
<i>Garter snake</i> (Herptile)	85%	0%	5%	0%	5%	5	1.0E+00	0.023	0.27
<i>Red fox</i> (Pred. Mammal)	20%	10%	40%	15%	10%	250	1.9E-01	0.23	4.9
<i>Red-tailed hawk</i> (Pred. Bird)	5%	5%	55%	10%	20%	500	9.6E-02	0.23	1.5

NOTES:

- [a] Bioaccumulation data presented in: Appendix Q, Table Q-1  
 [b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.  
 [c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2  
 [d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-3

## ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## FINAL CREEK, BADGER ARMY AMMUNITION PLANT

## EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
SO4	2.6E+02
PB	4.0E+01
24DNT	6.0E+00
26DNT	4.0E+01
NIT	1.1E+01
SN	6.3E+01
DEP	1.3E-01
DNBP	2.6E+01
DPA	1.5E+01
2NDPA	2.0E+00
NC	7.4E+02
NH3	1.8E+03

## ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert BAF [a]	Tissue Level (mg/kg)	Plant BAF [a]	Tissue Level (mg/kg)
5.0E-02	1.3E+01	5.0E-02	1.3E+01
2.4E+00	9.7E+01	2.0E-01	8.0E+00
1.0E+00	6.0E+00	1.0E+00	6.0E+00
1.0E+00	4.0E+01	1.0E+00	4.0E+01
5.0E-02	5.5E-01	5.0E-02	5.5E-01
1.0E+00	6.3E+01	1.0E+00	6.3E+01
1.0E+00	1.3E-01	5.3E-01	6.9E-02
1.0E+00	2.6E+01	6.5E-02	1.7E+00
1.0E+00	1.5E+01	1.0E+00	1.5E+01
1.0E+00	2.0E+00	6.0E-01	1.2E+00
5.0E-02	3.7E+01	5.0E-02	3.7E+01
5.0E-02	9.0E+01	5.0E-02	9.0E+01

## BAF VALUES FOR OTHER PREY ITEM

Small Mammal BAF	Small Bird BAF	Herptile BAF
0.0E+00	0.0E+00	0.0E+00
4.3E-01	3.8E-01	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
0.0E+00	0.0E+00	0.0E+00

SITE AREA: 2.00 acres

10 Nov 92

BAFCAAC.wkl

TABLE R-3  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
FINAL CREEK, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]					
CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
SO4	4.5E+01	2.8E+00	2.0E+00	7.9E-01	2.2E+00
PB	1.5E+02	8.4E+00	7.4E+00	2.0E+00	5.1E+00
24DNT	1.1E+01	6.6E-01	5.0E-01	2.4E-01	7.5E-01
26DNT	7.0E+01	4.4E+00	3.3E+00	1.6E+00	5.0E+00
NIT	1.9E+00	1.2E-01	8.7E-02	3.4E-02	9.3E-02
SN	1.1E+02	6.9E+00	5.2E+00	2.5E+00	7.9E+00
DEP	2.2E-01	1.3E-02	1.1E-02	4.8E-03	1.5E-02
DNBP	4.2E+01	2.3E+00	2.1E+00	8.7E-01	2.8E+00
DPA	2.6E+01	1.6E+00	1.2E+00	6.0E-01	1.9E+00
2NDPA	3.4E+00	2.0E-01	1.6E-01	7.4E-02	2.4E-01
NC	1.3E+02	7.9E+00	5.8E+00	2.3E+00	6.2E+00
NH3	3.1E+02	1.9E+01	1.4E+01	5.5E+00	1.5E+01

TABLE R-3

## ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## FINAL CREEK, BADGER ARMY AMMUNITION PLANT

## EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet					Home Range (acres)	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Herpeto-fauna	Birds				
<i>Short-tailed shrew</i>	85%	10%	0%	0%	0%	1.3	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i>	75%	20%	0%	0%	0%	5	4.0E-01	0.0095	0.087
<i>Garter snake</i>	85%	0%	5%	0%	5%	5	4.0E-01	0.023	0.27
<i>Red fox</i>	20%	10%	40%	15%	10%	250	8.0E-03	0.23	4.9
<i>Red-tailed hawk</i>	5%	5%	55%	10%	20%	500	4.0E-03	0.23	1.5

## NOTES:

[a] Bioaccumulation data presented in: Appendix Q, Table Q-1

[b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.

[c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2

[d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-4  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
FINAL CREEK, BADGER ARMY AMMUNITION PLANT

EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
SO4	2.6E+02
PB	4.0E+01
24DNT	6.0E+00
26DNT	4.0E+01
NIT	1.1E+01
SN	6.3E+01
DEP	1.3E-01
DNBP	2.6E+01
DPA	1.5E+01
2NDPA	2.0E+00
NC	7.4E+02
NH3	1.8E+03

ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert BAF [a]	Tissue Level (mg/kg)	Plant BAF [a]	Tissue Level (mg/kg)
5.0E-02	1.3E+01	5.0E-02	1.3E+01
2.4E+00	9.7E+01	2.0E-01	8.0E+00
1.0E+00	6.0E+00	1.0E+00	6.0E+00
1.0E+00	4.0E+01	1.0E+00	4.0E+01
5.0E-02	5.5E-01	5.0E-02	5.5E-01
1.0E+00	6.3E+01	1.0E+00	6.3E+01
1.0E+00	1.3E-01	5.3E-01	6.9E-02
1.0E+00	2.6E+01	6.5E-02	1.7E+00
1.0E+00	1.5E+01	1.0E+00	1.5E+01
1.0E+00	2.0E+00	6.0E-01	1.2E+00
5.0E-02	3.7E+01	5.0E-02	3.7E+01
5.0E-02	9.0E+01	5.0E-02	9.0E+01

BAF VALUES FOR OTHER PREY ITEM

Small Mammal BAF	Small Bird BAF	Herpetic BAF
0.0E+00	0.0E+00	0.0E+00
4.3E-01	3.8E-01	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
0.0E+00	0.0E+00	0.0E+00

SITE AREA: 2.00 acres

TABLE R-4  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
FINAL CREEK, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]					
CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
SO4	4.5E+01	1.1E+00	8.2E-01	6.3E-03	8.8E-03
PB	1.5E+02	3.3E+00	3.0E+00	1.6E-02	2.0E-02
24DNT	1.1E+01	2.6E-01	2.0E-01	1.9E-03	3.0E-03
26DNT	7.0E+01	1.7E+00	1.3E+00	1.3E-02	2.0E-02
INIT	1.9E+00	4.7E-02	3.5E-02	2.7E-04	3.7E-04
SN	1.1E+02	2.8E+00	2.1E+00	2.0E-02	3.2E-02
DEP	2.2E-01	5.1E-03	4.3E-03	3.8E-05	6.1E-05
DNBP	4.2E+01	9.2E-01	8.5E-01	6.9E-03	1.1E-02
DPA	2.6E+01	6.6E-01	5.0E-01	4.8E-03	7.5E-03
2NDPA	3.4E+00	8.0E-02	6.6E-02	5.9E-04	9.4E-04
NC	1.3E+02	3.2E+00	2.3E+00	1.8E-02	2.5E-02
NH3	3.1E+02	7.7E+00	5.7E+00	4.4E-02	6.1E-02



TABLE R-4  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
FINAL CREEK, BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet					Soil	Home Range (acres)	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Herpeto-fauna	Birds					
<i>Short-tailed shrew</i>	(Small Mammal) 85%	10%	0%	0%	0%	5%	1.3	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i>	(Small Bird) 75%	20%	0%	0%	0%	5%	5	4.0E-01	0.0095	0.087
<i>Garter snake</i>	(Herptile) 85%	0%	5%	0%	5%	5%	5	4.0E-01	0.023	0.27
<i>Red fox</i>	(Pred. Mammal) 20%	10%	40%	15%	10%	5%	250	8.0E-03	0.23	4.9
<i>Red-tailed hawk</i>	(Pred. Bird) 5%	5%	55%	10%	20%	5%	500	4.0E-03	0.23	1.5

NOTES:

- [a] Bioaccumulation data presented in: Appendix Q, Table Q-1  
 [b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.  
 [c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2  
 [d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

**TABLE R-5**  
**ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION**

# REMEDIAL INVESTIGATION SETTLING POND 1, BADGER ARMY AMMUNITION PLANT

EXPOSURE CONCENTRATION DATA	
CHEMICAL	CONCENTRATION (mg/kg)
SO4	2.5E+03
PB	1.8E+02
24DNT	1.7E+02
26DNT	2.6E+01
NIT.	1.3E+01
SN	5.7E+01
DEP	4.6E+02
DNBP	1.4E+01
DPA	1.0E+01
2NDPA	9.7E-01
NC	6.0E+04
NH3	7.4E+02

ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS				
Invert	Tissue		Plant	Tissue Level (mg/kg)
	BAF [a]	Level (mg/kg)		
5.0E-02	1.3E+02	5.0E-02	1.3E+02	
2.4E+00	4.4E+02	2.0E-01	3.6E+01	
1.0E+00	1.7E+02	1.0E+00	1.7E+02	
1.0E+00	2.6E+01	1.0E+00	2.6E+01	
5.0E-02	6.5E-01	5.0E-02	6.5E-01	
1.0E+00	5.7E+01	1.0E+00	5.7E+01	
1.0E+00	4.6E+02	5.3E-01	2.5E+02	
1.0E+00	1.4E+01	6.5E-02	9.1E-01	
1.0E+00	1.0E+01	1.0E+00	1.0E+01	
1.0E+00	9.7E-01	6.0E-01	5.8E-01	
5.0E-02	3.0E+03	5.0E-02	3.0E+03	
5.0E-02	3.7E+01	5.0E-02	3.7E+01	

BAF VALUES FOR OTHER PREY ITEM					
	Small Mammal		Small Bird		Herpetile BAF
	BAF		BAF		
	0.0E+00		0.0E+00		0.0E+00
	4.3E-01		3.8E-01		1.0E+00
	1.0E+00		1.0E+00		1.0E+00
	1.0E+00		1.0E+00		1.0E+00
	0.0E+00		0.0E+00		0.0E+00
	1.0E+00		1.0E+00		1.0E+00
	1.0E+00		1.0E+00		1.0E+00
	1.0E+00		1.0E+00		1.0E+00
	1.0E+00		1.0E+00		1.0E+00
	0.0E+00		0.0E+00		0.0E+00
	0.0E+00		0.0E+00		0.0E+00

**SITE AREA:** 24.00 acres

TABLE R-5  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SETTLING POND 1, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]					
CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
SO4	4.3E+02	2.7E+01	2.0E+01	7.6E+00	2.1E+01
PB	6.8E+02	3.8E+01	3.4E+01	1.1E+01	2.9E+01
24DNT	3.0E+02	1.9E+01	1.5E+01	8.1E+00	2.6E+01
26DNT	4.6E+01	2.8E+00	2.2E+00	1.2E+00	4.0E+00
NIT	2.2E+00	1.4E-01	1.0E-01	4.0E-02	1.1E-01
SN	1.0E+02	6.2E+00	4.9E+00	2.7E+00	8.7E+00
DEP	7.7E+02	4.6E+01	3.9E+01	2.0E+01	6.6E+01
DNBP	2.2E+01	1.2E+00	1.2E+00	5.6E-01	1.9E+00
DPA	1.8E+01	1.1E+00	8.5E-01	4.7E-01	1.5E+00
2NDPA	1.6E+00	9.7E-02	8.2E-02	4.3E-02	1.4E-01
NC	1.0E+04	6.4E+02	4.7E+02	1.8E+02	5.1E+02
NH3	1.3E+02	7.9E+00	5.8E+00	2.3E+00	6.2E+00

TABLE R-5  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SETTLING POND 1, BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet					Home Range (acres)	Soil	Birds	Herpeto-fauna	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Herpeto-fauna	Birds							
<i>Short-tailed shrew</i> (Small Mammal)	85%	10%	0%	0%	0%	1.3	5%	0%	0%	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i> (Small Bird)	75%	20%	0%	0%	0%	5	5%	0%	0%	1.0E+00	0.0095	0.087
<i>Garter snake</i> (Herptile)	85%	0%	5%	0%	5%	5	5%	5%	0%	1.0E+00	0.023	0.27
<i>Red fox</i> (Pred. Mammal)	20%	10%	40%	15%	10%	250	5%	10%	15%	9.6E-02	0.23	4.9
<i>Red-tailed hawk</i> (Pred. Bird)	5%	5%	55%	10%	20%	500	5%	20%	10%	4.8E-02	0.23	1.5

NOTES:

- [a] Bioaccumulation data presented in: Appendix Q, Table Q-1  
 [b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.  
 [c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2  
 [d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-6

ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SETTLING POND 1, BADGER ARMY AMMUNITION PLANT

## EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
SO4	2.5E+03
PB	1.8E+02
24DNT	1.7E+02
26DNT	2.6E+01
NIT	1.3E+01
SN	5.7E+01
DEP	4.6E+02
DNRP	1.4E+01
DPA	1.0E+01
2NDPA	9.7E-01
NC	6.0E+04
NH3	7.4E+02

## ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert BAF [a]	Tissue		Plant BAF [a]	Tissue Level (mg/kg)
	Level (mg/kg)	Level (mg/kg)		
5.0E-02	1.3E+02	1.3E+02	5.0E-02	1.3E+02
2.4E+00	4.4E+02	3.6E+01	2.0E-01	3.6E+01
1.0E+00	1.7E+02	1.7E+02	1.0E+00	1.7E+02
1.0E+00	2.6E+01	2.6E+01	1.0E+00	2.6E+01
5.0E-02	6.5E-01	6.5E-01	5.0E-02	6.5E-01
1.0E+00	5.7E+01	5.7E+01	1.0E+00	5.7E+01
1.0E+00	4.6E+02	2.5E+02	5.3E-01	2.5E+02
1.0E+00	1.4E+01	9.1E-01	6.5E-02	9.1E-01
1.0E+00	1.0E+01	1.0E+01	1.0E+00	1.0E+01
1.0E+00	9.7E-01	5.8E-01	6.0E-01	5.8E-01
5.0E-02	3.0E+03	3.0E+03	5.0E-02	3.0E+03
5.0E-02	3.7E+01	3.7E+01	5.0E-02	3.7E+01

## BAF VALUES FOR OTHER PREY ITEM

Small Mammal BAF	Small		Herptile BAF
	Bird BAF	Bird BAF	
0.0E+00	0.0E+00	0.0E+00	0.0E+00
4.3E-01	3.8E-01	3.8E-01	1.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00	0.0E+00
0.0E+00	0.0E+00	0.0E+00	0.0E+00

SITE AREA:

24.00 acres

06-Nov-92

BA SPICR.wkl

TABLE R-6  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SETTLING POND 1, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]

CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
SO4	4.3E+02	2.7E+01	2.0E+01	7.3E-01	1.0E+00
PB	6.8E+02	3.8E+01	3.4E+01	1.1E+00	1.4E+00
24DNT	3.0E+02	1.9E+01	1.5E+01	7.8E-01	1.3E+00
26DNT	4.6E+01	2.8E+00	2.2E+00	1.2E-01	1.9E-01
NIT	2.2E+00	1.4E-01	1.0E-01	3.8E-03	5.3E-03
SN	1.0E+02	6.2E+00	4.9E+00	2.6E-01	4.2E-01
DEP	7.7E+02	4.6E+01	3.9E+01	1.9E+00	3.2E+00
DNBP	2.2E+01	1.2E+00	1.2E+00	5.4E-02	8.9E-02
DPA	1.8E+01	1.1E+00	8.5E-01	4.5E-02	7.4E-02
2NDPA	1.6E+00	9.7E-02	8.2E-02	4.1E-03	6.7E-03
NC	1.0E+04	6.4E+02	4.7E+02	1.8E+01	2.4E+01
NH3	1.3E+02	7.9E+00	5.8E+00	2.2E-01	3.0E-01

TABLE R-6  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SETTLING POND 1, BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet					Home Range (acres)	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Herpeto-fauna	Birds				
<i>Short-tailed shrew</i> (Small Mammal)	85%	10%	0%	0%	0%	1.3	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i> (Small Bird)	75%	20%	0%	0%	0%	5	1.0E+00	0.0095	0.087
<i>Garter snake</i> (Herpetile)	85%	0%	5%	0%	5%	5	1.0E+00	0.023	0.27
<i>Red fox</i> (Pred. Mammal)	20%	10%	40%	15%	10%	250	9.6E-02	0.23	4.9
<i>Red-tailed hawk</i> (Pred. Bird)	5%	5%	55%	10%	20%	500	4.8E-02	0.23	1.5

NOTES:

- [a] Bioaccumulation data presented in: Appendix Q, Table Q-1  
 [b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.  
 [c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2  
 [d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-7  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SETTLING POND 2, BADGER ARMY AMMUNITION PLANT

EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
SO4	6.4E+01
PB	2.5E+02
24DNT	7.6E+00
NIT	4.3E+01
SN	5.3E+01
DEP	1.4E+02
DNBP	7.4E-01
DPA	1.5E+00
NC	2.8E+02
NH3	8.4E+02

ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert BAF [e]	Tissue		Plant BAF [e]	Tissue Level (mg/kg)
	Level (mg/kg)	Level		
5.0E-02	3.2E+00		5.0E-02	3.2E+00
2.4E+00	6.1E+02		2.0E-01	5.0E+01
1.0E+00	7.6E+00		1.0E+00	7.6E+00
5.0E-02	2.2E+00		5.0E-02	2.2E+00
1.0E+00	5.3E+01		1.0E+00	5.3E+01
1.0E+00	1.4E+02		5.3E-01	7.2E+01
1.0E+00	7.4E-01		6.5E-02	4.8E-02
1.0E+00	1.5E+00		1.0E+00	1.5E+00
5.0E-02	1.4E+01		5.0E-02	1.4E+01
5.0E-02	4.2E+01		5.0E-02	4.2E+01

BAF VALUES FOR OTHER PREY ITEM

Small Mammal BAF	Small		Herptile BAF
	Bird BAF	Bird BAF	
0.0E+00	0.0E+00		0.0E+00
4.3E-01	3.8E-01		1.0E+00
1.0E+00	1.0E+00		1.0E+00
0.0E+00	0.0E+00		0.0E+00
1.0E+00	1.0E+00		1.0E+00
1.0E+00	1.0E+00		1.0E+00
1.0E+00	1.0E+00		1.0E+00
1.0E+00	1.0E+00		1.0E+00
0.0E+00	0.0E+00		0.0E+00
0.0E+00	0.0E+00		0.0E+00

SITE AREA: 7.00 acres



TABLE R-7  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SETTLING POND 2, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]

CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
SO4	1.1E+01	6.8E-01	5.0E-01	2.0E-01	5.4E-01
PB	9.4E+02	5.2E+01	4.7E+01	1.6E+01	4.0E+01
24DNT	1.3E+01	8.3E-01	6.5E-01	3.6E-01	1.2E+00
NIT	7.4E+00	4.6E-01	3.4E-01	1.3E-01	3.6E-01
SN	9.3E+01	5.8E+00	4.5E+00	2.5E+00	8.1E+00
DEP	2.3E+02	1.3E+01	1.1E+01	5.9E+00	1.9E+01
DNBP	1.2E+00	6.6E-02	6.2E-02	2.9E-02	9.8E-02
DPA	2.6E+00	1.6E-01	1.3E-01	7.0E-02	2.3E-01
NC	4.8E+01	3.0E+00	2.2E+00	8.5E-01	2.4E+00
NH3	1.4E+02	8.9E+00	6.6E+00	2.6E+00	7.1E+00

TABLE R-7  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SETTLING POND 2, BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet					Home Range (acres)	Soil	Birds	Herpeto-fauna	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverte	Plants	Small Mammals	Small	Herpeto-fauna							
Short-tailed shrew	(Small Mammal)	85%	10%	0%	0%	0%	5%	0%	0%	1.0E+00	0.037	0.021
Eastern meadowlark	(Small Bird)	75%	20%	0%	0%	0%	5%	0%	0%	1.0E+00	0.0095	0.087
Garter snake	(Herptile)	85%	0%	5%	0%	5%	5%	5%	0%	1.0E+00	0.023	0.27
Red fox	(Pred. Mammal)	20%	10%	40%	15%	10%	5%	10%	15%	2.8E-02	0.23	4.9
Red-tailed hawk	(Pred. Bird)	5%	5%	55%	10%	20%	5%	20%	10%	1.4E-02	0.23	1.5

NOTES:

- [a] Bioaccumulation data presented in: Appendix Q, Table Q-1  
 [b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.  
 [c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2  
 [d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-8  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SETTLING POND 2, BADGER ARMY AMMUNITION PLANT

EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
SO4	6.4E+01
PB	2.5E+02
24DNT	7.6E+00
NIT	4.3E+01
SN	5.3E+01
DEP	1.4E+02
DNBP	7.4E-01
DPA	1.5E+00
NC	2.8E+02
NH3	8.4E+02

ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert	BAF [a]	Tissue Level (mg/kg)	Plant BAF [a]	Tissue Level (mg/kg)
5.0E-02	5.0E-02	3.2E+00	5.0E-02	3.2E+00
2.4E+00	6.1E+02	6.1E+02	2.0E-01	5.0E+01
1.0E+00	7.6E+00	7.6E+00	1.0E+00	7.6E+00
5.0E-02	2.2E+00	2.2E+00	5.0E-02	2.2E+00
1.0E+00	5.3E+01	5.3E+01	1.0E+00	5.3E+01
1.0E+00	1.4E+02	1.4E+02	5.3E-01	7.2E+01
1.0E+00	7.4E-01	7.4E-01	6.5E-02	4.8E-02
1.0E+00	1.5E+00	1.5E+00	1.0E+00	1.5E+00
5.0E-02	1.4E+01	1.4E+01	5.0E-02	1.4E+01
5.0E-02	4.2E+01	4.2E+01	5.0E-02	4.2E+01

BAF VALUES FOR OTHER PREY ITEM

Small Mammal BAF	Small Bird BAF	Herptile BAF
0.0E+00	0.0E+00	0.0E+00
4.3E-01	3.8E-01	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
0.0E+00	0.0E+00	0.0E+00

SITE AREA: 7.00 acres

TABLE R-8

## ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SETTLING POND 2, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]

CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
SO <sub>4</sub>	1.1E+01	6.8E-01	5.0E-01	5.5E-03	7.6E-03
PB	9.4E+02	5.2E+01	4.7E+01	4.4E-01	5.6E-01
24DNT	1.3E+01	8.3E-01	6.5E-01	1.0E-02	1.6E-02
NIT	7.4E+00	4.6E-01	3.4E-01	3.7E-03	5.1E-03
SN	9.3E+01	5.8E+00	4.5E+00	7.0E-02	1.1E-01
DEP	2.3E+02	1.3E+01	1.1E+01	1.6E-01	2.7E-01
DNBP	1.2E+00	6.6E-02	6.2E-02	8.3E-04	1.4E-03
DPA	2.6E+00	1.6E-01	1.3E-01	2.0E-03	3.2E-03
NC	4.8E+01	3.0E+00	2.2E+00	2.4E-02	3.3E-02
NH <sub>3</sub>	1.4E+02	8.9E+00	6.6E+00	7.2E-02	9.9E-02

TABLE R-8  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SETTLING POND 2, BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet					Home Range (acres)	Soil	Birds	Herpeto-fauna	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Herpeto-fauna	Birds							
<i>Short-tailed shrew</i>	(Small Mammal)	85%	10%	0%	0%	1.3	5%	0%	0%	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i>	(Small Bird)	75%	20%	0%	0%	5	5%	0%	0%	1.0E+00	0.0095	0.087
<i>Garter snake</i>	(Herpille)	85%	0%	5%	0%	5	5%	5%	5%	1.0E+00	0.023	0.27
<i>Red fox</i>	(Pred. Mammal)	20%	10%	40%	15%	250	5%	10%	10%	2.8E-02	0.23	4.9
<i>Red-tailed hawk</i>	(Pred. Bird)	5%	5%	55%	10%	500	5%	20%	20%	1.4E-02	0.23	1.5

NOTES:

- [a] Bioaccumulation data presented in: Appendix Q, Table Q-1  
 [b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.  
 [c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2  
 [d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-9

## ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SETTLING POND 3, BADGER ARMY AMMUNITION PLANT

## EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
SO4	3.6E+01
PB	3.4E+01
24DNT	2.6E+00
26DNT	1.5E+00
NIT	4.9E+00
SN	7.2E+01
DEP	4.4E+01
DNBP	1.7E+01
DPA	2.8E+00
NC	1.9E+02
NH3	5.2E+02

## ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert BAF [a]	Tissue		Plant BAF [a]	Tissue Level (mg/kg)
	Level (mg/kg)	Level (mg/kg)		
5.0E-02	1.8E+00	1.8E+00	5.0E-02	1.8E+00
2.4E+00	8.3E+01	6.8E+00	2.0E-01	6.8E+00
1.0E+00	2.6E+00	2.6E+00	1.0E+00	2.6E+00
1.0E+00	1.5E+00	1.5E+00	1.0E+00	1.5E+00
5.0E-02	2.5E-01	2.5E-01	5.0E-02	2.5E-01
1.0E+00	7.2E+01	7.2E+01	1.0E+00	7.2E+01
1.0E+00	4.4E+01	2.3E+01	5.3E-01	2.3E+01
1.0E+00	1.7E+01	1.1E+00	6.5E-02	1.1E+00
1.0E+00	2.8E+00	2.8E+00	1.0E+00	2.8E+00
5.0E-02	9.5E+00	9.5E+00	5.0E-02	9.5E+00
5.0E-02	2.6E+01	2.6E+01	5.0E-02	2.6E+01

## BAF VALUES FOR OTHER PREY ITEM

Small Mammal BAF	Small		Small Mammal BAF	Small Bird BAF	Herpetile BAF
	Level (mg/kg)	Level (mg/kg)			
0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00
4.3E-01	3.8E-01	3.8E-01	4.3E-01	3.8E-01	1.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00
0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00

SITE AREA: 25.00 acres

10-Nov-92

BA SP3AC v1.1

TABLE R-9  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SETTLING POND 3, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]					
CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
SO4	6.2E+00	3.8E-01	2.8E-01	1.1E-01	3.0E-01
PB	1.3E+02	7.1E+00	6.4E+00	2.1E+00	5.5E+00
24DNT	4.6E+00	2.8E-01	2.2E-01	1.2E-01	4.0E-01
26DNT	2.6E+00	1.6E-01	1.3E-01	7.0E-02	2.3E-01
NIT	8.4E-01	5.2E-02	3.9E-02	1.5E-02	4.1E-02
SN	1.3E+02	7.9E+00	6.1E+00	3.4E+00	1.1E+01
DEP	7.4E+01	4.4E+00	3.7E+00	1.9E+00	6.3E+00
DNBP	2.8E+01	1.5E+00	1.5E+00	6.9E-01	2.3E+00
DPA	4.9E+00	3.1E-01	2.4E-01	1.3E-01	4.3E-01
NC	3.3E+01	2.0E+00	1.5E+00	5.8E-01	1.6E+00
NH3	8.9E+01	5.5E+00	4.1E+00	1.6E+00	4.4E+00

TABLE R-9  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SETTLING POND 3, BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet				Soil	Home Range (acres)	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Herpeto-fauna					
<i>Short-tailed shrew</i>	(Small Mammal)	85%	10%	0%	0%	1.3	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i>	(Small Bird)	75%	20%	0%	0%	5	1.0E+00	0.0095	0.087
<i>Garter snake</i>	(Herptile)	85%	0%	5%	0%	5	1.0E+00	0.023	0.27
<i>Red fox</i>	(Pred. Mammal)	20%	10%	40%	15%	250	1.0E-01	0.23	4.9
<i>Red-tailed hawk</i>	(Pred. Bird)	5%	5%	55%	10%	500	5.0E-02	0.23	1.5

NOTES:

- [a] Bioaccumulation data presented in: Appendix Q, Table Q-1  
 [b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.  
 [c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2  
 [d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)



TABLE R-10  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SETTLING POND 3, BADGER ARMY AMMUNITION PLANT

EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
SO4	3.6E+01
PB	3.4E+01
24DNT	2.6E+00
26DNT	1.5E+00
NIT	4.9E+00
SN	7.2E+01
DEP	4.4E+01
DNBP	1.7E+01
DPA	2.8E+00
NC	1.9E+02
NH3	5.2E+02

ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert BAF [a]	Tissue Level (mg/kg)	Plant BAF [a]	Tissue Level (mg/kg)
5.0E-02	1.8E+00	5.0E-02	1.8E+00
2.4E+00	8.3E+01	2.0E-01	6.8E+00
1.0E+00	2.6E+00	1.0E+00	2.6E+00
1.0E+00	1.5E+00	1.0E+00	1.5E+00
5.0E-02	2.5E-01	5.0E-02	2.5E-01
1.0E+00	7.2E+01	1.0E+00	7.2E+01
1.0E+00	4.4E+01	5.3E-01	2.3E+01
1.0E+00	1.7E+01	6.5E-02	1.1E+00
1.0E+00	2.8E+00	1.0E+00	2.8E+00
5.0E-02	9.5E+00	5.0E-02	9.5E+00
5.0E-02	2.6E+01	5.0E-02	2.6E+01

BAF VALUES FOR OTHER PREY ITEM

Small Mammal BAF	Small Bird BAF	Herptile BAF
0.0E+00	0.0E+00	0.0E+00
4.3E-01	3.8E-01	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
0.0E+00	0.0E+00	0.0E+00

SITE AREA: 25.00 acres

TABLE R-10

## ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SETTLING POND 3, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]

CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
SO4	6.2E+00	3.8E-01	2.8E-01	1.1E-02	1.5E-02
PB	1.3E+02	7.1E+00	6.4E+00	2.1E-01	2.7E-01
24DNT	4.6E+00	2.8E-01	2.2E-01	1.2E-02	2.0E-02
26DNT	2.6E+00	1.6E-01	1.3E-01	7.0E-03	1.2E-02
NIT	8.4E-01	5.2E-02	3.9E-02	1.5E-03	2.1E-03
SN	1.3E+02	7.9E+00	6.1E+00	3.4E-01	5.5E-01
DEP	7.4E+01	4.4E+00	3.7E+00	1.9E-01	3.1E-01
DNBP	2.8E+01	1.5E+00	1.5E+00	6.9E-02	1.2E-01
DPA	4.9E+00	3.1E-01	2.4E-01	1.3E-02	2.1E-02
NC	3.3E+01	2.0E+00	1.5E+00	5.8E-02	8.0E-02
NH3	8.9E+01	5.5E+00	4.1E+00	1.6E-01	2.2E-01

TABLE R-10  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SETTLING POND 3, BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet				Home Range (acres)	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Herpeto-fauna				
<i>Short-tailed shrew</i> (Small Mammal)	85%	10%	0%	0%	1.3	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i> (Small Bird)	75%	20%	0%	0%	5	1.0E+00	0.0095	0.087
<i>Garter snake</i> (Herptile)	85%	0%	5%	0%	5	1.0E+00	0.023	0.27
<i>Red fox</i> (Pred. Mammal)	20%	10%	40%	15%	250	1.0E-01	0.23	4.9
<i>Red-tailed hawk</i> (Pred. Bird)	5%	5%	55%	10%	500	5.0E-02	0.23	1.5

NOTES:

- [a] Bioaccumulation data presented in: Appendix Q, Table Q-1  
 [b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.  
 [c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2  
 [d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-11

ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

SETTLING POND 4, BADGER ARMY AMMUNITION PLANT

## EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
SO4	4.0E+02
PB	3.0E+02
AL	6.0E+04
NIT	1.0E+01
SN	7.7E+01
DPA	3.6E-01
NC	1.0E+03
NH3	9.6E+02

## ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert BAF [a]	Tissue Level (mg/kg)	Plant BAF [a]	Tissue Level (mg/kg)
5.0E-02	2.0E+01	5.0E-02	2.0E+01
2.4E+00	7.3E+02	2.0E-01	6.0E+01
1.0E+00	6.0E+04	1.0E+00	6.0E+04
5.0E-02	5.0E-01	5.0E-02	5.0E-01
1.0E+00	7.7E+01	1.0E+00	7.7E+01
1.0E+00	3.6E-01	1.0E+00	3.6E-01
5.0E-02	5.2E+01	5.0E-02	5.2E+01
5.0E-02	4.8E+01	5.0E-02	4.8E+01

## BAF VALUES FOR OTHER PREY ITEM

Small Mammal BAF	Small Bird BAF	Herpille BAF
0.0E+00	0.0E+00	0.0E+00
4.3E-01	3.8E-01	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
0.0E+00	0.0E+00	0.0E+00

SITE AREA:

20.00 acres

10-Nov-92

BA SP4AC w.t.1

TABLE R-11  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SETTLING POND 4, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]

CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
SO4	6.9E+01	4.3E+00	3.2E+00	1.2E+00	3.4E+00
PB	1.1E+03	6.3E+01	5.6E+01	1.9E+01	4.8E+01
AL	1.1E+05	6.6E+03	5.1E+03	2.8E+03	9.2E+03
NIT	1.7E+00	1.1E-01	7.9E-02	3.1E-02	8.4E-02
SN	1.4E+02	8.4E+00	6.6E+00	3.6E+00	1.2E+01
DPA	6.3E-01	3.9E-02	3.1E-02	1.7E-02	5.5E-02
NC	1.8E+02	1.1E+01	8.2E+00	3.2E+00	8.8E+00
NH3	1.6E+02	1.0E+01	7.6E+00	2.9E+00	8.1E+00

TABLE R-11  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SETTLING POND 4, BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet					Home Range (acres)	Soil	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Herpeto-fauna	Birds					
<i>Short-tailed shrew</i>	(Small Mammal) 85%	10%	0%	0%	0%	1.3	5%	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i>	(Small Bird) 75%	20%	0%	0%	0%	5	5%	1.0E+00	0.0095	0.087
<i>Garter snake</i>	(Herpetile) 85%	0%	5%	0%	5%	5	5%	1.0E+00	0.023	0.27
<i>Red fox</i>	(Pred. Mammal) 20%	10%	40%	15%	10%	250	5%	8.0E-02	0.23	4.9
<i>Red-tailed hawk</i>	(Pred. Bird) 5%	5%	55%	10%	20%	500	5%	4.0E-02	0.23	1.5

NOTES:

- (a) Bioaccumulation data presented in: Appendix Q, Table Q-1  
 (b) Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.  
 (c) Documentation of exposure parameters presented in: Appendix Q, Table Q-2  
 (d) Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-12

## ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SETTLING POND 4, BADGER ARMY AMMUNITION PLANT

## EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
SO4	4.0E+02
PB	3.0E+02
AL	6.0E+04
NIT	1.0E+01
SN	7.7E+01
DPA	3.6E-01
NC	1.0E+03
NH3	9.6E+02

## ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert BAF [a]	Tissue Level (mg/kg)	Plant BAF [a]	Tissue Level (mg/kg)
5.0E-02	2.0E+01	5.0E-02	2.0E+01
2.4E+00	7.3E+02	2.0E-01	6.0E+01
1.0E+00	6.0E+04	1.0E+00	6.0E+04
5.0E-02	5.0E-01	5.0E-02	5.0E-01
1.0E+00	7.7E+01	1.0E+00	7.7E+01
1.0E+00	3.6E-01	1.0E+00	3.6E-01
5.0E-02	5.2E+01	5.0E-02	5.2E+01
5.0E-02	4.8E+01	5.0E-02	4.8E+01

## BAF VALUES FOR OTHER PREY ITEM

Small Mammal BAF	Small Bird BAF	Herptile BAF
0.0E+00	0.0E+00	0.0E+00
4.3E-01	3.8E-01	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
0.0E+00	0.0E+00	0.0E+00

SITE AREA: 20.00 acres

TABLE R-12  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SETTLING POND 4, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]					
CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
SO4	6.9E+01	4.3E+00	3.2E+00	9.8E-02	1.3E-01
PB	1.1E+03	6.3E+01	5.6E+01	1.5E+00	1.9E+00
AL	1.1E+05	6.6E+03	5.1E+03	2.3E+02	3.7E+02
NIT	1.7E+00	1.1E-01	7.9E-02	2.4E-03	3.4E-03
SN	1.4E+02	8.4E+00	6.6E+00	2.9E-01	4.7E-01
DPA	6.3E-01	3.9E-02	3.1E-02	1.4E-03	2.2E-03
NC	1.8E+02	1.1E+01	8.2E+00	2.5E-01	3.5E-01
NH3	1.6E+02	1.0E+01	7.6E+00	2.3E-01	3.2E-01



TABLE R-12  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SETTLING POND 4, BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet				Home Range (acres)	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Herpeto-fauna				
<i>Short-tailed shrew</i> (Small Mammal)	85%	10%	0%	0%	1.3	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i> (Small Bird)	75%	20%	0%	0%	5	1.0E+00	0.0095	0.087
<i>Garler snake</i> (Herptile)	85%	0%	5%	0%	5	1.0E+00	0.023	0.27
<i>Red fox</i> (Pred. Mammal)	20%	10%	40%	15%	250	8.0E-02	0.23	4.9
<i>Red-tailed hawk</i> (Pred. Bird)	5%	5%	55%	10%	500	4.0E-02	0.23	1.5

NOTES:

- [a] Bioaccumulation data presented in: Appendix Q, Table Q-1  
 [b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.  
 [c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2  
 [d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-13

## ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SPOILS DISPOSAL AREA 1, BADGER ARMY AMMUNITION PLANT

## EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
SO4	1.5E+02
PB	3.5E+02
24DNT	1.2E+01
26DNT	1.0E+00
NIT	1.6E+01
SN	3.7E+00
ZN	2.1E+02
DNBP	5.1E+01
DPA	2.4E+01
NC	1.1E+04
CH2Cl2	1.0E-02
B2EHP	3.5E-01
NG	1.9E+01
CL	1.9E+01
BR	1.2E+01
DNOP	8.6E+00

## ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert	Tissue Level (mg/kg)	Plant	Tissue Level (mg/kg)
BAF [a]		BAF [a]	
5.0E-02	7.3E+00	5.0E-02	7.3E+00
2.4E+00	8.5E+02	2.0E-01	7.0E+01
1.0E+00	1.2E+01	1.0E+00	1.2E+01
1.0E+00	1.0E+00	1.0E+00	1.0E+00
5.0E-02	8.0E-01	5.0E-02	8.0E-01
1.0E+00	3.7E+00	1.0E+00	3.7E+00
7.3E+00	1.5E+03	1.0E+01	2.1E+03
1.0E+00	5.1E+01	6.5E-02	3.3E+00
1.0E+00	2.4E+01	1.0E+00	2.4E+01
5.0E-02	5.5E+02	5.0E-02	5.5E+02
1.0E+00	1.0E-02	1.0E+00	1.0E-02
1.0E+00	3.5E-01	4.3E-02	1.5E-02
1.0E+00	1.9E+01	1.0E+00	1.9E+01
5.0E-02	9.5E-01	5.0E-02	9.5E-01
5.0E-02	6.0E-01	5.0E-02	6.0E-01
1.0E+00	8.6E+00	4.3E-02	3.7E-01

## BAF VALUES FOR OTHER PREY ITEM

Small Mammal BAF	Small Bird BAF	Herptile BAF
0.0E+00	0.0E+00	0.0E+00
4.3E-01	3.8E-01	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00
5.1E+00	1.0E+01	1.0E+01
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00

SITE AREA: 5.00 acres

10-Nov-92

BA SDIAC w.k.l

TABLE R-13  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 1, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]						
CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk	
SO4	2.5E+01	1.6E+00	1.2E+00	4.5E-01	1.2E+00	
PB	1.3E+03	7.3E+01	6.5E+01	2.2E+01	5.6E+01	
24DNT	2.1E+01	1.3E+00	1.0E+00	5.6E-01	1.8E+00	
26DNT	1.8E+00	1.1E-01	8.5E-02	4.7E-02	1.5E-01	
NIT	2.7E+00	1.7E-01	1.3E-01	4.9E-02	1.3E-01	
SN	6.5E+00	4.0E-01	3.1E-01	1.7E-01	5.6E-01	
ZN	2.7E+03	1.7E+02	2.1E+02	4.2E+02	1.6E+03	
DNBP	8.1E+01	4.5E+00	4.3E+00	2.0E+00	6.7E+00	
DPA	4.2E+01	2.6E+00	2.0E+00	1.1E+00	3.7E+00	
NC	1.9E+03	1.2E+02	8.7E+01	3.4E+01	9.3E+01	
CH2Cl2	1.8E-02	1.1E-03	8.5E-04	4.7E-04	1.5E-03	
B2EHP	5.6E-01	3.1E-02	2.9E-02	1.4E-02	4.6E-02	
NG	3.3E+01	2.1E+00	1.6E+00	8.9E-01	2.9E+00	
CL	3.3E+00	2.0E-01	1.5E-01	5.8E-02	1.6E-01	
BR	2.1E+00	1.3E-01	9.5E-02	3.7E-02	1.0E-01	
DNOP	1.4E+01	7.6E-01	7.2E-01	3.4E-01	1.1E+00	

TABLE R-13

## ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SPOILS DISPOSAL AREA 1, BADGER ARMY AMMUNITION PLANT

## EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet					Home Range (acres)	Soil	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Herpeto-fauna	Birds					
<i>Short-tailed shrew</i> (Small Mammal)	85%	10%	0%	0%	0%	1.3	5%	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i> (Small Bird)	75%	20%	0%	0%	0%	5	5%	1.0E+00	0.0095	0.087
<i>Garter snake</i> (Herptile)	85%	0%	5%	0%	5%	5	5%	1.0E+00	0.023	0.27
<i>Red fox</i> (Pred. Mammal)	20%	10%	40%	15%	10%	250	5%	2.0E-02	0.23	4.9
<i>Red-tailed hawk</i> (Pred. Bird)	5%	5%	55%	10%	20%	500	5%	1.0E-02	0.23	1.5

## NOTES:

[a] Bioaccumulation data presented in: Appendix Q, Table Q-1

[b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.

[c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2

[d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

**TABLE R-14**  
**ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION**

**REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA I, BADGER ARMY AMMUNITION PLANT**

EXPOSURE CONCENTRATION DATA	
CHEMICAL	CONCENTRATION (mg/kg)
SO4	1.5E+02
PB	3.5E+02
24DNT	1.2E+01
26DNT	1.0E+00
NIT	1.6E+01
SN	3.7E+00
ZN	2.1E+02
DNBP	5.1E+01
DPA	2.4E+01
NC	1.1E+04
CH2Cl2	1.0E-02
B2EHP	3.5E-01
NG	1.9E+01
CL	1.9E+01
BR	1.2E+01
DNOP	8.6E+00

ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS			
Invert	Tissue		Tissue Level (mg/kg)
	BAF [a]	Level (mg/kg)	
5.0E-02	7.3E+00	5.0E-02	7.3E+00
2.4E+00	8.5E+02	2.0E-01	7.0E+01
1.0E+00	1.2E+01	1.0E+00	1.2E+01
1.0E+00	1.0E+00	1.0E+00	1.0E+00
5.0E-02	8.0E-01	5.0E-02	8.0E-01
1.0E+00	3.7E+00	1.0E+00	3.7E+00
7.3E+00	1.5E+03	1.0E+01	2.1E+03
1.0E+00	5.1E+01	6.5E-02	3.3E+00
1.0E+00	2.4E+01	1.0E+00	2.4E+01
5.0E-02	5.5E+02	5.0E-02	5.5E+02
1.0E+00	1.0E-02	1.0E+00	1.0E-02
1.0E+00	3.5E-01	4.3E-02	1.5E-02
1.0E+00	1.9E+01	1.0E+00	1.9E+01
5.0E-02	9.5E-01	5.0E-02	9.5E-01
5.0E-02	6.0E-01	5.0E-02	6.0E-01
1.0E+00	8.6E+00	4.3E-02	3.7E-01

BAF VALUES FOR OTHER PREY ITEM	Small		Small		Herptile BAF
	Mammal BAF	Bird BAF	Bird BAF	Herptile BAF	
	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00
	4.3E-01	3.8E-01	1.0E+00	1.0E+00	1.0E+00
	1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00
	1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00
	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00
	1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00
	5.1E+00	1.0E+01	1.0E+01	1.0E+01	1.0E+01
	1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00
	1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00
	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00
	1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00
	1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00
	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00
	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00
	1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00

**SITE AREA:** 5.00 acres

TABLE R-14

## ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SPOILS DISPOSAL AREA 1, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]

CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
SO4	2.5E+01	1.6E+00	1.2E+00	8.9E-03	1.2E-02
PB	1.3E+03	7.3E+01	6.5E+01	4.3E-01	5.6E-01
24DNT	2.1E+01	1.3E+00	1.0E+00	1.1E-02	1.8E-02
26DNT	1.8E+00	1.1E-01	8.5E-02	9.4E-04	1.5E-03
NIT	2.7E+00	1.7E-01	1.3E-01	9.8E-04	1.3E-03
SN	6.5E+00	4.0E-01	3.1E-01	3.5E-03	5.6E-03
ZN	2.7E+03	1.7E+02	2.1E+02	8.5E+00	1.6E+01
DNBP	8.1E+01	4.5E+00	4.3E+00	4.1E-02	6.7E-02
DPA	4.2E+01	2.6E+00	2.0E+00	2.3E-02	3.7E-02
NC	1.9E+03	1.2E+02	8.7E+01	6.7E-01	9.3E-01
CH2Cl2	1.8E-02	1.1E-03	8.5E-04	9.4E-06	1.5E-05
B2EHP	5.6E-01	3.1E-02	2.9E-02	2.8E-04	4.6E-04
NG	3.3E+01	2.1E+00	1.6E+00	1.8E-02	2.9E-02
CL	3.3E+00	2.0E-01	1.5E-01	1.2E-03	1.6E-03
BR	2.1E+00	1.3E-01	9.5E-02	7.3E-04	1.0E-03
DNOP	1.4E+01	7.6E-01	7.2E-01	6.8E-03	1.1E-02

TABLE R-14  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 1, BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet					Soil	Home Range (acres)	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Herpeto-fauna	Birds					
<i>Short-tailed shrew</i>	(Small Mammal) 85%	10%	0%	0%	0%	5%	1.3	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i>	(Small Bird) 75%	20%	0%	0%	0%	5%	5	1.0E+00	0.0095	0.087
<i>Garter snake</i>	(Herpitle) 85%	0%	5%	0%	5%	5%	5	1.0E+00	0.023	0.27
<i>Red fox</i>	(Pred. Mammal) 20%	10%	40%	15%	10%	5%	250	2.0E-02	0.23	4.9
<i>Red-tailed hawk</i>	(Pred. Bird) 5%	5%	55%	10%	20%	5%	500	1.0E-02	0.23	1.5

NOTES:

- [a] Bioaccumulation data presented in: Appendix Q, Table Q-1  
 [b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.  
 [c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2  
 [d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-15

## ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SPOILS DISPOSAL AREA 2, BADGER ARMY AMMUNITION PLANT

## EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
SO4	1.3E+02
PB	3.7E+02
NIT	1.0E+01
SN	4.0E+00
ZN	7.5E+02
DNBP	5.8E+00
DPA	3.2E+00
NC	8.0E+03
CH2Cl2	1.2E-02
BR	4.0E+00
CL	2.3E+01
24DNT	1.3E+00

## ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert BAF [a]	Tissue Level (mg/kg)	Plant BAF [a]	Tissue Level (mg/kg)
5.0E-02	6.5E+00	5.0E-02	6.5E+00
2.4E+00	9.1E+02	2.0E-01	7.5E+01
5.0E-02	5.0E-01	5.0E-02	5.0E-01
1.0E+00	4.0E+00	1.0E+00	4.0E+00
7.3E+00	5.5E+03	1.0E+01	7.5E+03
1.0E+00	5.8E+00	6.5E-02	3.8E-01
1.0E+00	3.2E+00	1.0E+00	3.2E+00
5.0E-02	4.0E+02	5.0E-02	4.0E+02
1.0E+00	1.2E-02	1.0E+00	1.2E-02
5.0E-02	2.0E-01	5.0E-02	2.0E-01
5.0E-02	1.2E+00	5.0E-02	1.2E+00
1.0E+00	1.3E+00	1.0E+00	1.3E+00

## BAF VALUES FOR OTHER PREY ITEM

Small Mammal BAF	Small Bird BAF	Herpille BAF
0.0E+00	0.0E+00	0.0E+00
4.3E-01	3.8E-01	1.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00
5.1E+00	1.0E+01	1.0E+01
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
0.0E+00	0.0E+00	0.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00

SITE AREA: 3.50 acres

10-Nov-92

BA SD2AC.wkl



TABLE R-15  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 2, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kg BW-day) [b]					
CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
SO4	2.2E+01	1.4E+00	1.0E+00	4.0E-01	1.1E+00
PB	1.4E+03	7.8E+01	6.9E+01	2.1E+01	5.4E+01
NIT	1.7E+00	1.1E-01	7.9E-02	3.1E-02	8.4E-02
SN	7.1E+00	4.4E-01	3.4E-01	1.8E-01	5.6E-01
ZN	9.6E+03	6.2E+02	6.9E+02	1.2E+03	4.5E+03
DNBP	9.3E+00	5.1E-01	4.8E-01	2.1E-01	7.0E-01
DPA	5.6E+00	3.5E-01	2.7E-01	1.4E-01	4.5E-01
NC	1.4E+03	8.5E+01	6.3E+01	2.4E+01	6.7E+01
CH2Cl2	2.1E-02	1.3E-03	1.0E-03	5.2E-04	1.7E-03
BR	6.9E-01	4.3E-02	3.2E-02	1.2E-02	3.4E-02
CL	4.0E+00	2.4E-01	1.8E-01	7.0E-02	1.9E-01
24DNT	2.3E+00	1.4E-01	1.1E-01	5.6E-02	1.8E-01

TABLE R-15  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 2, BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet					Home Range (acres)	Soil	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverte	Plants	Small Mammals	Herpeto-fauna	Birds					
<i>Short-tailed shrew</i> (Small Mammal)	85%	10%	0%	0%	0%	1.3	5%	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i> (Small Bird)	75%	20%	0%	0%	0%	5	5%	7.0E-01	0.0095	0.087
<i>Garter snake</i> (Herptile)	85%	0%	5%	0%	5%	5	5%	7.0E-01	0.023	0.27
<i>Red fox</i> (Pred. Mammal)	20%	10%	40%	15%	10%	250	5%	1.4E-02	0.23	4.9
<i>Red-tailed hawk</i> (Pred. Bird)	5%	5%	55%	10%	20%	500	5%	7.0E-03	0.23	1.5

NOTES:

- [a] Bioaccumulation data presented in: Appendix Q, Table Q-1  
 [b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.  
 [c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2  
 [d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-16  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 2, BADGER ARMY AMMUNITION PLANT

EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
SO4	1.3E+02
PB	3.7E+02
NIT	1.0E+01
SN	4.0E+00
ZN	7.5E+02
DNBP	5.8E+00
DPA	3.2E+00
NC	8.0E+03
CH2Cl2	1.2E-02
BR	4.0E+00
CL	2.3E+01
24DNT	1.3E+00

ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert BAF [a]	Tissue Level (mg/kg)	Plant BAF [a]	Tissue Level (mg/kg)
5.0E-02	6.5E+00	5.0E-02	6.5E+00
2.4E+00	9.1E+02	2.0E-01	7.5E+01
5.0E-02	5.0E-01	5.0E-02	5.0E-01
1.0E+00	4.0E+00	1.0E+00	4.0E+00
7.3E+00	5.5E+03	1.0E+01	7.5E+03
1.0E+00	5.8E+00	6.5E-02	3.8E-01
1.0E+00	3.2E+00	1.0E+00	3.2E+00
5.0E-02	4.0E+02	5.0E-02	4.0E+02
1.0E+00	1.2E-02	1.0E+00	1.2E-02
5.0E-02	2.0E-01	5.0E-02	2.0E-01
5.0E-02	1.2E+00	5.0E-02	1.2E+00
1.0E+00	1.3E+00	1.0E+00	1.3E+00

BAF VALUES FOR OTHER PREY ITEM

Small Mammal BAF	Small Bird BAF	Herp BAF
0.0E+00	0.0E+00	0.0E+00
4.3E-01	3.8E-01	1.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00
5.1E+00	1.0E+01	1.0E+01
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00

SITE AREA: 3.50 acres

TABLE R-16  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 2, BADGER ARMY AMMUNITION PLANT  
TOTAL BODY DOSE (mg/kgBW-day) [b]

CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
SO4	2.2E+01	9.7E-01	7.2E-01	5.6E-03	7.7E-03
PB	1.4E+03	5.5E+01	4.9E+01	3.0E-01	3.8E-01
NIT	1.7E+00	7.5E-02	5.5E-02	4.3E-04	5.9E-04
SN	7.1E+00	3.1E-01	2.4E-01	2.5E-03	3.9E-03
ZN	9.6E+03	4.3E+02	4.8E+02	1.7E+01	3.2E+01
DNBP	9.3E+00	3.6E-01	3.4E-01	3.0E-03	4.9E-03
DPA	5.6E+00	2.4E-01	1.9E-01	1.9E-03	3.1E-03
NC	1.4E+03	6.0E+01	4.4E+01	3.4E-01	4.7E-01
CH2Cl2	2.1E-02	9.2E-04	7.0E-04	7.3E-06	1.2E-05
BR	6.9E-01	3.0E-02	2.2E-02	1.7E-04	2.4E-04
CL	4.0E+00	1.7E-01	1.3E-01	9.8E-04	1.4E-03
24DNT	2.3E+00	9.9E-02	7.6E-02	7.9E-04	1.3E-03

TABLE R-16  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 2, BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet					Soil	Home Range (acres)	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Herpeto-fauna	Birds					
<i>Short-tailed shrew</i> (Small Mammal)	85%	10%	0%	0%	0%	5%	1.3	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i> (Small Bird)	75%	20%	0%	0%	0%	5%	5	7.0E-01	0.0095	0.087
<i>Garter snake</i> (Herpitle)	85%	0%	5%	0%	5%	5%	5	7.0E-01	0.023	0.27
<i>Red fox</i> (Pred. Mammal)	20%	10%	40%	15%	10%	5%	250	1.4E-02	0.23	4.9
<i>Red-tailed hawk</i> (Pred. Bird)	5%	5%	55%	10%	20%	5%	500	7.0E-03	0.23	1.5

NOTES:

- [a] Bioaccumulation data presented in: Appendix Q, Table Q-1  
 [b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.  
 [c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2  
 [d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-17

ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

SPOILS DISPOSAL AREA 3, BADGER ARMY AMMUNITION PLANT

## EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
SO4	7.5E+01
PB	6.7E+01
NIT	2.2E+01
SN	5.8E+00
ZN	2.5E+02
DNBP	4.0E+00
DPA	2.2E+00
NC	3.8E+03
CH2Cl2	2.5E-02
CL	1.7E+01
24DNT	1.1E+00

## ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert BAF [a]	Tissue		Plant BAF [a]	Tissue Level (mg/kg)
	Level (mg/kg)	Level (mg/kg)		
5.0E-02	3.8E+00	3.8E+00	5.0E-02	3.8E+00
2.4E+00	1.6E+02	1.3E+01	2.0E-01	1.3E+01
5.0E-02	1.1E+00	1.1E+00	5.0E-02	1.1E+00
1.0E+00	5.8E+00	5.8E+00	1.0E+00	5.8E+00
7.3E+00	1.8E+03	2.5E+03	1.0E+01	2.5E+03
1.0E+00	4.0E+00	2.6E-01	6.5E-02	2.6E-01
1.0E+00	2.2E+00	2.2E+00	1.0E+00	2.2E+00
5.0E-02	1.9E+02	1.9E+02	5.0E-02	1.9E+02
1.0E+00	2.5E-02	2.5E-02	1.0E+00	2.5E-02
5.0E-02	8.5E-01	8.5E-01	5.0E-02	8.5E-01
1.0E+00	1.1E+00	1.1E+00	1.0E+00	1.1E+00

## BAF VALUES FOR OTHER PREY ITEM

Small Mammal BAF	Small		Small Bird BAF	Herpetile BAF
	BAF	BAF		
0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00
4.3E-01	3.8E-01	3.8E-01	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00
5.1E+00	1.0E+01	1.0E+01	1.0E+01	1.0E+01
1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00	1.0E+00

SITE AREA:

3.00 acres

10-Nov-92

BA SD3AC w.f.1

TABLE R-17  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 3, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]					
CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
SO4	1.3E+01	8.0E-01	5.9E-01	2.3E-01	6.3E-01
PB	2.5E+02	1.4E+01	1.2E+01	3.7E+00	9.3E+00
NIT	3.8E+00	2.3E-01	1.7E-01	6.7E-02	1.9E-01
SN	1.0E+01	6.3E-01	4.8E-01	2.4E-01	7.8E-01
ZN	3.2E+03	2.1E+02	2.2E+02	3.7E+02	1.4E+03
DNBP	6.4E+00	3.6E-01	3.3E-01	1.4E-01	4.6E-01
DPA	3.9E+00	2.4E-01	1.8E-01	9.3E-02	3.0E-01
NC	6.5E+02	4.0E+01	3.0E+01	1.2E+01	3.2E+01
CH2Cl2	4.4E-02	2.7E-03	2.1E-03	1.1E-03	3.4E-03
CL	2.9E+00	1.8E-01	1.3E-01	5.2E-02	1.4E-01
24DNT	1.9E+00	1.2E-01	9.2E-02	4.6E-02	1.5E-01

TABLE R-17  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 3, BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet					Home Range (acres)	Soil	Birds	Herpeto-fauna	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Small	Herpeto-fauna							
<i>Short-tailed shrew</i> (Small Mammal)	85%	10%	0%	0%	0%	1.3	5%	0%	0%	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i> (Small Bird)	75%	20%	0%	0%	0%	5	5%	0%	0%	6.0E-01	0.0095	0.087
<i>Garter snake</i> (Herptile)	85%	0%	5%	5%	0%	5	5%	5%	0%	6.0E-01	0.023	0.27
<i>Red fox</i> (Pred. Mammal)	20%	10%	40%	15%	10%	250	5%	10%	15%	1.2E-02	0.23	4.9
<i>Red-tailed hawk</i> (Pred. Bird)	5%	5%	55%	10%	10%	500	5%	20%	10%	6.0E-03	0.23	1.5

NOTES:

- [a] Bioaccumulation data presented in: Appendix Q, Table Q-1  
 [b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.  
 [c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2  
 [d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)



TABLE R-18

ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

SPOILS DISPOSAL AREA 3, BADGER ARMY AMMUNITION PLANT

## EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
SO4	7.5E+01
PB	6.7E+01
NIT	2.2E+01
SN	5.8E+00
ZN	2.5E+02
DNBP	4.0E+00
DPA	2.2E+00
NC	3.8E+03
CH2Cl2	2.5E-02
CL	1.7E+01
24DNT	1.1E+00

## ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert	Tissue		Plant	Tissue Level (mg/kg)
	BAF [a]	Level (mg/kg)		
5.0E-02	3.8E+00	3.8E+00	5.0E-02	3.8E+00
2.4E+00	1.6E+02	1.3E+01	2.0E-01	1.3E+01
5.0E-02	1.1E+00	1.1E+00	5.0E-02	1.1E+00
1.0E+00	5.8E+00	5.8E+00	1.0E+00	5.8E+00
7.3E+00	1.8E+03	2.5E+03	1.0E+01	2.5E+03
1.0E+00	4.0E+00	2.6E-01	6.5E-02	2.6E-01
1.0E+00	2.2E+00	2.2E+00	1.0E+00	2.2E+00
5.0E-02	1.9E+02	1.9E+02	5.0E-02	1.9E+02
1.0E+00	2.5E-02	2.5E-02	1.0E+00	2.5E-02
5.0E-02	8.5E-01	8.5E-01	5.0E-02	8.5E-01
1.0E+00	1.1E+00	1.1E+00	1.0E+00	1.1E+00

## BAF VALUES FOR OTHER PREY ITEM

Small Mammal	Small Bird		Herp.ile
	BAF	BAF	BAF
0.0E+00	0.0E+00	0.0E+00	0.0E+00
4.3E-01	3.8E-01	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00
5.1E+00	1.0E+01	1.0E+01	1.0E+01
1.0E+00	1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00

SITE AREA: 3.00 acres

10-Nov-92

BA\_SDCR.wkt

TABLE R-18

ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

SPOILS DISPOSAL AREA 3, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]

CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
SO4	1.3E+01	4.8E-01	3.5E-01	2.7E-03	3.8E-03
PB	2.5E+02	8.4E+00	7.5E+00	4.4E-02	5.6E-02
NIT	3.8E+00	1.4E-01	1.0E-01	8.1E-04	1.1E-03
SN	1.0E+01	3.8E-01	2.9E-01	2.9E-03	4.7E-03
ZN	3.2E+03	1.2E+02	1.3E+02	4.4E+00	8.4E+00
DNBP	6.4E+00	2.1E-01	2.0E-01	1.7E-03	2.8E-03
DPA	3.9E+00	1.4E-01	1.1E-01	1.1E-03	1.8E-03
NC	6.5E+02	2.4E+01	1.8E+01	1.4E-01	1.9E-01
CH2Cl2	4.4E-02	1.6E-03	1.3E-03	1.3E-05	2.0E-05
CL	2.9E+00	1.1E-01	8.0E-02	6.2E-04	8.6E-04
24DNT	1.9E+00	7.2E-02	5.5E-02	5.6E-04	8.9E-04

TABLE R-18  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 3, BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet					Home Range (acres)	Soil	Birds	Herpeto-fauna	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Small	Herpeto-fauna							
<i>Short-tailed shrew</i>	(Small Mammal)	85%	10%	0%	0%	1.3	5%	0%	0%	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i>	(Small Bird)	75%	20%	0%	0%	5	5%	0%	0%	6.0E-01	0.0095	0.087
<i>Garter snake</i>	(Herptile)	85%	0%	5%	0%	5	5%	5%	5%	6.0E-01	0.023	0.27
<i>Red fox</i>	(Pred. Mammal)	20%	10%	40%	15%	250	5%	10%	15%	1.2E-02	0.23	4.9
<i>Red-tailed hawk</i>	(Pred. Bird)	5%	5%	55%	10%	500	5%	20%	10%	6.0E-03	0.23	1.5

NOTES:

- [a] Bioaccumulation data presented in: Appendix Q, Table Q-1  
 [b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.  
 [c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2  
 [d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-19

## ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SPOILS DISPOSAL AREA 4, BADGER ARMY AMMUNITION PLANT

## EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
SO4	1.4E+02
PB	1.2E+02
24DNT	7.0E-01
NIT	1.2E+01
SN	1.6E+00
ZN	2.0E+02
DNBP	4.4E+00
DPA	1.1E+00
NC	3.0E+03
CH2Cl2	1.0E-02
B2EHP	3.2E-01
CL	1.3E+01
DNOP	6.3E-01

## ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert	Tissue Level (mg/kg)	Plant	Tissue Level (mg/kg)
BAF [e]		BAF [e]	
5.0E-02	7.0E+00	5.0E-02	7.0E+00
2.4E+00	2.9E+02	2.0E-01	2.4E+01
1.0E+00	7.0E-01	1.0E+00	7.0E-01
5.0E-02	6.0E-01	5.0E-02	6.0E-01
1.0E+00	1.6E+00	1.0E+00	1.6E+00
7.3E+00	1.5E+03	1.0E+01	2.0E+03
1.0E+00	4.4E+00	6.5E-02	2.9E-01
1.0E+00	1.1E+00	1.0E+00	1.1E+00
5.0E-02	1.5E+02	5.0E-02	1.5E+02
1.0E+00	1.0E-02	1.0E+00	1.0E-02
1.0E+00	3.2E-01	4.3E-02	1.4E-02
5.0E-02	6.5E-01	5.0E-02	6.5E-01
1.0E+00	6.3E-01	4.3E-02	2.7E-02

## BAF VALUES FOR OTHER PREY ITEM

Small Mammal BAF	Small Bird BAF	Herptile BAF
0.0E+00	0.0E+00	0.0E+00
4.3E-01	3.8E-01	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00
5.1E+00	1.0E+01	1.0E+01
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00

SITE AREA: 5.00 acres

10-Nov-92

BA\_SD4AC wkl

TABLE R-19  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 4, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
SO4	2.4E+01	1.5E+00	1.1E+00	4.2E-01	1.2E+00
PB	4.5E+02	2.5E+01	2.2E+01	7.5E+00	1.9E+01
24DNT	1.2E+00	7.6E-02	6.0E-02	3.3E-02	1.1E-01
NIT	2.1E+00	1.3E-01	9.5E-02	3.7E-02	1.0E-01
SN	2.9E+00	1.8E-01	1.4E-01	7.7E-02	2.5E-01
ZN	2.6E+03	1.7E+02	2.1E+02	4.1E+02	1.5E+03
DNBP	7.0E+00	3.9E-01	3.7E-01	1.8E-01	5.8E-01
DPA	1.9E+00	1.2E-01	9.4E-02	5.2E-02	1.7E-01
NC	5.2E+02	3.2E+01	2.4E+01	9.2E+00	2.5E+01
CH2Cl2	1.8E-02	1.1E-03	8.5E-04	4.7E-04	1.5E-03
B2EHP	5.1E-01	2.8E-02	2.7E-02	1.3E-02	4.2E-02
CL	2.2E+00	1.4E-01	1.0E-01	4.0E-02	1.1E-01
DNOP	1.0E+00	5.6E-02	5.3E-02	2.5E-02	8.3E-02

TOTAL BODY DOSE (mg/kgBW-day) [b]

TABLE R-19  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 4, BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet					Home Range (acres)	Soil	Birds	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Herpeto-fauna							
<i>Short-tailed shrew</i> (Small Mammal)	85%	10%	0%	0%	0%	1.3	5%	0%	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i> (Small Bird)	75%	20%	0%	0%	0%	5	5%	0%	1.0E+00	0.0095	0.087
<i>Garter snake</i> (Herptile)	85%	0%	5%	0%	5%	5	5%	5%	1.0E+00	0.023	0.27
<i>Red fox</i> (Pred. Mammal)	20%	10%	40%	15%	10%	250	5%	10%	2.0E-02	0.23	4.9
<i>Red-tailed hawk</i> (Pred. Bird)	5%	5%	55%	10%	20%	500	5%	20%	1.0E-02	0.23	1.5

NOTES:

- [a] Bioaccumulation data presented in: Appendix Q, Table Q-1  
 [b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.  
 [c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2  
 [d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-20  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 4, BADGER ARMY AMMUNITION PLANT

EXPOSURE CONCENTRATION DATA		ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS				BAF VALUES FOR OTHER PREY ITEM			
CHEMICAL	CONCENTRATION (mg/kg)	Invert BAF [a]	Tissue Level (mg/kg)	Plant BAF [a]	Tissue Level (mg/kg)	Small Mammal BAF	Small Bird BAF	Herptile BAF	
SO4	1.4E+02	5.0E-02	7.0E+00	5.0E-02	7.0E+00	0.0E+00	0.0E+00	0.0E+00	
PB	1.2E+02	2.4E+00	2.9E+02	01	2.4E+01	4.3E-01	3.8E-01	1.0E+00	
24DNT	7.0E-01	1.0E+00	7.0E-01	0E+00	7.0E-01	1.0E+00	1.0E+00	1.0E+00	
NIT	1.2E+01	5.0E-02	6.0E-01	5.0E-02	6.0E-01	0.0E+00	0.0E+00	0.0E+00	
SN	1.6E+00	1.0E+00	1.6E+00	1.0E-01	1.6E+00	1.0E+00	1.0E+00	1.0E+00	
ZN	2.0E+02	7.3E+00	1.5E+03	1.0E+01	2.0E+03	5.1E+00	1.0E+01	1.0E+01	
DNBP	4.4E+00	1.0E+00	4.4E+00	6.5E-02	2.9E-01	1.0E+00	1.0E+00	1.0E+00	
DPA	1.1E+00	1.0E+00	1.1E+00	1.0E+00	1.1E+00	1.0E+00	1.0E+00	1.0E+00	
NC	3.0E+03	5.0E-02	1.5E+02	5.0E-02	1.5E+02	0.0E+00	0.0E+00	0.0E+00	
CH2C12	1.0E-02	1.0E+00	1.0E-02	1.0E+00	1.0E-02	1.0E+00	1.0E+00	1.0E+00	
B2EHP	3.2E-01	1.0E+00	3.2E-01	4.3E-02	1.4E-02	1.0E+00	1.0E+00	1.0E+00	
CL	1.3E+01	5.0E-02	6.5E-01	5.0E-02	6.5E-01	0.0E+00	0.0E+00	0.0E+00	
DNOP	6.3E-01	1.0E+00	6.3E-01	4.3E-02	2.7E-02	1.0E+00	1.0E+00	1.0E+00	

SITE AREA: 5.00 acres

TABLE R-20  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 4, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]					
CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
SO4	2.4E+01	1.5E+00	1.1E+00	8.5E-03	1.2E-02
PB	4.5E+02	2.5E+01	2.2E+01	1.5E-01	1.9E-01
24DNT	1.2E+00	7.6E-02	6.0E-02	6.6E-04	1.1E-03
NIT	2.1E+00	1.3E-01	9.5E-02	7.3E-04	1.0E-03
SN	2.9E+00	1.8E-01	1.4E-01	1.5E-03	2.5E-03
ZN	2.6E+03	1.7E+02	2.1E+02	8.2E+00	1.5E+01
DNBP	7.0E+00	3.9E-01	3.7E-01	3.5E-03	5.8E-03
DPA	1.9E+00	1.2E-01	9.4E-02	1.0E-03	1.7E-03
NC	5.2E+02	3.2E+01	2.4E+01	1.8E-01	2.5E-01
CH2Cl2	1.8E-02	1.1E-03	8.5E-04	9.4E-06	1.5E-05
B2EHP	5.1E-01	2.8E-02	2.7E-02	2.5E-04	4.2E-04
CL	2.2E+00	1.4E-01	1.0E-01	7.9E-04	1.1E-03
DNOP	1.0E+00	5.6E-02	5.3E-02	5.0E-04	8.3E-04



TABLE R-20  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 4, BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet				Home Range (acres)	Soil	Birds	Herpeto-fauna	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Herpeto-fauna							
Short-tailed shrew (Small Mammal)	85%	10%	0%	0%	1.3	5%	0%	0%	1.0E+00	0.037	0.021
Eastern meadowlark (Small Bird)	75%	20%	0%	0%	5	5%	0%	0%	1.0E+00	0.0095	0.087
Garter snake (Herpitle)	85%	0%	5%	0%	5	5%	5%	0%	1.0E+00	0.023	0.27
Red fox (Pred. Mammal)	20%	10%	40%	15%	250	5%	10%	15%	2.0E-02	0.23	4.9
Red-tailed hawk (Pred. Bird)	5%	5%	55%	10%	500	5%	20%	10%	1.0E-02	0.23	1.5

NOTES:

- [a] Bioaccumulation data presented in: Appendix Q, Table Q-1  
 [b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.  
 [c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2  
 [d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-21

ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

SPOILS DISPOSAL AREA 5, BADGER ARMY AMMUNITION PLANT

## EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
SO4	3.8E+01
PB	1.0E+02
NIT	1.8E+01
SN	1.9E+00
ZN	3.1E+02
DNBP	6.5E+00
DPA	2.4E+00
NC	1.1E+04
CH2Cl2	1.0E-02
BR	1.6E+01
CL	1.8E+01
DNOP	2.0E-01

## ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert	Tissue		Plant	Tissue Level (mg/kg)
	BAF [a]	Level (mg/kg)	BAF [a]	
5.0E-02	1.9E+00	5.0E-02	1.9E+00	1.9E+00
2.4E+00	2.5E+02	2.0E-01	2.0E+01	2.0E+01
5.0E-02	9.0E-01	5.0E-02	9.0E-01	9.0E-01
1.0E+00	1.9E+00	1.0E+00	1.9E+00	1.9E+00
7.3E+00	2.2E+03	1.0E+01	3.1E+03	3.1E+03
1.0E+00	6.5E+00	6.5E-02	4.2E-01	4.2E-01
1.0E+00	2.4E+00	1.0E+00	2.4E+00	2.4E+00
5.0E-02	5.5E+02	5.0E-02	5.5E+02	5.5E+02
1.0E+00	1.0E-02	1.0E+00	1.0E-02	1.0E-02
5.0E-02	8.0E-01	5.0E-02	8.0E-01	8.0E-01
5.0E-02	9.0E-01	5.0E-02	9.0E-01	9.0E-01
1.0E+00	2.0E-01	4.3E-02	8.6E-03	8.6E-03

## BAF VALUES FOR OTHER PREY ITEM

Small Mammal	Small		Herptile
	BAF	Blrd BAF	BAF
0.0E+00	0.0E+00	0.0E+00	0.0E+00
4.3E-01	3.8E-01	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00
5.1E+00	1.0E+01	1.0E+01	1.0E+01
1.0E+00	1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00	0.0E+00
0.0E+00	0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00	1.0E+00

SITE AREA: 5.00 acres

10-Nov-92

BA SDSAC wkl

TABLE R-21  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 5, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]					
CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
SO4	6.5E+00	4.0E-01	3.0E-01	1.2E-01	3.2E-01
PB	3.8E+02	2.1E+01	1.9E+01	6.3E+00	1.6E+01
NIT	3.1E+00	1.9E-01	1.4E-01	5.5E-02	1.5E-01
SN	3.4E+00	2.1E-01	1.7E-01	9.1E-02	3.0E-01
ZN	3.9E+03	2.5E+02	3.1E+02	6.1E+02	2.3E+03
DNBP	1.0E+01	5.8E-01	5.5E-01	2.6E-01	8.6E-01
DPA	4.2E+00	2.6E-01	2.0E-01	1.1E-01	3.7E-01
NC	1.9E+03	1.2E+02	8.7E+01	3.4E+01	9.3E+01
CH2Cl2	1.8E-02	1.1E-03	8.5E-04	4.7E-04	1.5E-03
BR	2.7E+00	1.7E-01	1.3E-01	4.9E-02	1.3E-01
CL	3.1E+00	1.9E-01	1.4E-01	5.5E-02	1.5E-01
DNOP	3.2E-01	1.8E-02	1.7E-02	7.9E-03	2.6E-02

TABLE R-21  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

REMEDIATION INVESTIGATION  
SPOILS DISPOSAL AREA 5, BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet					Home Range (acres)	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Herpeto-fauna	Birds				
<i>Short-tailed shrew</i> (Small Mammal)	85%	10%	0%	0%	0%	1.3	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i> (Small Bird)	75%	20%	0%	0%	0%	5	1.0E+00	0.0095	0.087
<i>Garter snake</i> (Herpetile)	85%	0%	5%	0%	5%	5	1.0E+00	0.023	0.27
<i>Red fox</i> (Pred. Mammal)	20%	10%	40%	15%	10%	250	2.0E-02	0.23	4.9
<i>Red-tailed hawk</i> (Pred. Bird)	5%	5%	55%	10%	20%	500	1.0E-02	0.23	1.5

NOTES:

- [a] Bioaccumulation data presented in: Appendix Q, Table Q-1  
 [b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.  
 [c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2  
 [d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-22  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 5, BADGER ARMY AMMUNITION PLANT

EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
SO4	3.8E+01
PB	1.0E+02
NIT	1.8E+01
SN	1.9E+00
ZN	3.1E+02
DNBP	6.5E+00
DPA	2.4E+00
NC	1.1E+04
CH2Cl2	1.0E-02
BR	1.6E+01
CL	1.8E+01
DNOP	2.0E-01

ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert BAF [a]	Tissue Level (mg/kg)	Plant BAF [a]	Tissue Level (mg/kg)
5.0E-02	1.9E+00	5.0E-02	1.9E+00
2.4E+00	2.5E+02	2.0E-01	2.0E+01
5.0E-02	9.0E-01	5.0E-02	9.0E-01
1.0E+00	1.9E+00	1.0E+00	1.9E+00
7.3E+00	2.2E+03	1.0E+01	3.1E+03
1.0E+00	6.5E+00	6.5E-02	4.2E-01
1.0E+00	2.4E+00	1.0E+00	2.4E+00
5.0E-02	5.5E+02	5.0E-02	5.5E+02
1.0E+00	1.0E-02	1.0E+00	1.0E-02
5.0E-02	8.0E-01	5.0E-02	8.0E-01
5.0E-02	9.0E-01	5.0E-02	9.0E-01
1.0E+00	2.0E-01	4.3E-02	8.6E-03

BAF VALUES FOR OTHER PREY ITEM

Small Mammal BAF	Small Bird BAF	Herpetic BAF
0.0E+00	0.0E+00	0.0E+00
4.3E-01	3.8E-01	1.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00
5.1E+00	1.0E+01	1.0E+01
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
0.0E+00	0.0E+00	0.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00

SITE AREA: 5.00 acres

TABLE R-22  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 5, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]					
CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
SO4	6.5E+00	4.0E-01	3.0E-01	2.3E-03	3.2E-03
PB	3.8E+02	2.1E+01	1.9E+01	1.3E-01	1.6E-01
NIT	3.1E+00	1.9E-01	1.4E-01	1.1E-03	1.5E-03
SN	3.4E+00	2.1E-01	1.7E-01	1.8E-03	3.0E-03
ZN	3.9E+03	2.5E+02	3.1E+02	1.2E+01	2.3E+01
DNBP	1.0E+01	5.8E-01	5.5E-01	5.2E-03	8.6E-03
DPA	4.2E+00	2.6E-01	2.0E-01	2.3E-03	3.7E-03
NC	1.9E+03	1.2E+02	8.7E+01	6.7E-01	9.3E-01
CH2Cl2	1.8E-02	1.1E-03	8.5E-04	9.4E-06	1.5E-05
BR	2.7E+00	1.7E-01	1.3E-01	9.8E-04	1.3E-03
CL	3.1E+00	1.9E-01	1.4E-01	1.1E-03	1.5E-03
DNOP	3.2E-01	1.8E-02	1.7E-02	1.6E-04	2.6E-04

TABLE R-22

## ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SPOILS DISPOSAL AREA 5, BADGER ARMY AMMUNITION PLANT

## EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet					Home Range (acres)	Soil	Birds	Herpeto-fauna	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverte	Plants	Small Mammals	Small	Herpeto-fauna							
<i>Short-tailed shrew</i> (Small Mammal)	85%	10%	0%	0%	0%	1.3	5%	0%	0%	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i> (Small Bird)	75%	20%	0%	0%	0%	5	5%	0%	0%	1.0E+00	0.0095	0.087
<i>Garter snake</i> (Herptile)	85%	0%	5%	5%	0%	5	5%	5%	0%	1.0E+00	0.023	0.27
<i>Red fox</i> (Pred. Mammal)	20%	10%	40%	15%	10%	250	5%	10%	15%	2.0E-02	0.23	4.9
<i>Red-tailed hawk</i> (Pred. Bird)	5%	5%	55%	10%	20%	500	5%	20%	10%	1.0E-02	0.23	1.5

## NOTES:

(a) Bioaccumulation data presented in: Appendix Q, Table Q-1

(b) Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.

(c) Documentation of exposure parameters presented in: Appendix Q, Table Q-2

(d) Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-23

ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

ROCKET PASTE AREA, BADGER ARMY AMMUNITION PLANT

## EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
24DNT	8.1E+02
26DNT	3.3E+01
BAANTR	6.7E-01
CR	1.1E+02
DEP	5.0E+01
HG	7.2E-01
NG	1.5E+03
NNDPA	1.0E+04
PB	3.5E+03
PYR	9.3E-01
123PDA	1.9E+01
CHRY	1.0E+00
FANT	1.1E+00
NIT	1.2E+02
NNDMEA	3.0E-01
NNDNPA	2.3E-01
PHANTR	2.8E-01
SO4	2.3E+01

## ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert BAF [a]	Tissue Level (mg/kg)	Plant BAF [a]	Tissue Level (mg/kg)
1.0E+00	8.1E+02	1.0E+00	8.1E+02
1.0E+00	3.3E+01	1.0E+00	3.3E+01
1.0E+00	6.7E-01	2.2E-02	1.5E-02
1.6E-01	1.7E+01	1.0E-01	1.1E+01
1.0E+00	5.0E+01	5.3E-01	2.6E+01
3.4E-01	2.4E-01	1.0E+00	7.2E-01
1.0E+00	1.5E+03	1.0E+00	1.5E+03
1.0E+00	1.0E+04	6.0E-01	6.0E+03
2.4E+00	8.4E+03	2.0E-01	7.0E+02
1.0E+00	9.3E-01	5.9E-02	5.5E-02
1.0E+00	1.9E+01	1.0E+00	1.9E+01
1.0E+00	1.0E+00	2.2E-02	2.2E-02
1.0E+00	1.1E+00	5.7E-02	6.4E-02
5.0E-02	6.0E+00	5.0E-02	6.0E+00
1.0E+00	3.0E-01	1.0E+00	3.0E-01
1.0E+00	2.3E-01	1.0E+00	2.3E-01
1.0E+00	2.8E-01	1.0E-01	2.8E-02
5.0E-02	1.1E+00	5.0E-02	1.1E+00

## BAF VALUES FOR OTHER PREY ITEM

Small Mammal BAF	Small Bird BAF	Herptile BAF
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
5.0E+00	2.3E+00	1.0E+01
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
4.3E-01	3.8E-01	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00

SITE AREA: 606 acres

10-Nov-92

BA\_RPAAC.mtl



TABLE R-23  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
ROCKET PASTE AREA, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]					
CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
24DNT	1.4E+03	8.8E+01	6.9E+01	3.8E+01	1.2E+02
26DNT	5.7E+01	3.5E+00	2.8E+00	1.5E+00	5.0E+00
BAANTR	1.1E+00	5.8E-02	5.6E-02	2.6E-02	8.7E-02
CR	3.8E+01	2.3E+00	1.9E+00	1.1E+00	3.8E+00
DEP	8.4E+01	4.9E+00	4.2E+00	2.2E+00	7.1E+00
HG	5.5E-01	3.9E-02	3.1E-02	6.6E-02	2.3E-01
NG	2.6E+03	1.6E+02	1.3E+02	7.0E+01	2.3E+02
NNDPA	1.7E+04	1.0E+03	8.5E+02	4.4E+02	1.4E+03
PB	1.3E+04	7.2E+02	6.5E+02	2.2E+02	5.6E+02
PYR	1.5E+00	8.3E-02	7.8E-02	3.7E-02	1.2E-01
I23PDA	3.3E+01	2.1E+00	1.6E+00	8.9E-01	2.9E+00
CHRY	1.6E+00	8.8E-02	8.4E-02	3.9E-02	1.3E-01
FANT	1.8E+00	9.9E-02	9.4E-02	4.5E-02	1.5E-01
NIT	2.1E+01	1.3E+00	9.5E-01	3.7E-01	1.0E+00
NNDMEA	5.3E-01	3.3E-02	2.6E-02	1.4E-02	4.6E-02
NNDNPA	4.1E-01	2.5E-02	2.0E-02	1.1E-02	3.5E-02
PHANTR	4.5E-01	2.5E-02	2.3E-02	1.1E-02	3.7E-02
SO4	3.9E+00	2.4E-01	1.8E-01	7.0E-02	1.9E-01

TABLE R-23

## ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## ROCKET PASTE AREA, BADGER ARMY AMMUNITION PLANT

## EXPOSURE PARAMETERS [c]

Indicator Species		Percent Prey in Diet				Home Range (acres)	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
		Inverts	Plants	Small Mammals	Herpeto-fauna				
<i>Short-tailed shrew</i>	(Small Mammal)	85%	10%	0%	0%	1.3	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i>	(Small Bird)	75%	20%	0%	0%	5	1.0E+00	0.0095	0.087
<i>Garter snake</i>	(Herptile)	8.7%	0%	5%	0%	5	1.0E+00	0.023	0.27
<i>Red fox</i>	(Pred. Mammal)	20%	10%	40%	15%	250	1.0E+00	0.23	4.9
<i>Red-tailed hawk</i>	(Pred. Bird)	5%	5%	55%	10%	500	1.0E+00	0.23	1.5

## NOTES:

[a] Bioaccumulation data presented in: Appendix Q, Table Q-1

[b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.

[c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2

[d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-24

## ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## ROCKET PASTE AREA, BADGER ARMY AMMUNITION PLANT

## EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
24DNT	8.1E+02
26DNT	3.3E+01
BAANTR	6.7E-01
CR	1.1E+02
DEP	5.0E+01
HG	7.2E-01
NG	1.5E+03
NNDPA	1.0E+04
PB	3.5E+03
PYR	9.3E-01
123PDA	1.9E+01
CHRY	1.0E+00
FANT	1.1E+00
NIT	1.2E+02
NNDMEA	3.0E-01
NNDNPA	2.3E-01
PHANTR	2.8E-01
SO4	2.3E+01

## ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert BAF [a]	Tissue Level (mg/kg)	Plant BAF [a]	Tissue Level (mg/kg)
1.0E+00	8.1E+02	1.0E+00	8.1E+02
1.0E+00	3.3E+01	1.0E+00	3.3E+01
1.0E+00	6.7E-01	2.2E-02	1.5E-02
1.6E-01	1.7E+01	1.0E-01	1.1E+01
1.0E+00	5.0E+01	5.3E-01	2.6E+01
3.4E-01	2.4E-01	1.0E+00	7.2E-01
1.0E+00	1.5E+03	1.0E+00	1.5E+03
1.0E+00	1.0E+04	6.0E-01	6.0E+03
2.4E+00	8.4E+03	2.0E-01	7.0E+02
1.0E+00	9.3E-01	5.9E-02	5.5E-02
1.0E+00	1.9E+01	1.0E+00	1.9E+01
1.0E+00	1.0E+00	2.2E-02	2.2E-02
1.0E+00	1.1E+00	5.7E-02	6.4E-02
5.0E-02	6.0E+00	5.0E-02	6.0E+00
1.0E+00	3.0E-01	1.0E+00	3.0E-01
1.0E+00	2.3E-01	1.0E+00	2.3E-01
1.0E+00	2.8E-01	1.0E-01	2.8E-02
5.0E-02	1.1E+00	5.0E-02	1.1E+00

## BAF VALUES FOR OTHER PREY ITEM

Small Mammal BAF	Small Bird BAF	Herp BAF
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
5.0E+00	2.3E+00	1.0E+01
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
4.3E-01	3.8E-01	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00

SITE AREA: 606 acres

10-Nov-92

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TABLE R-24

## ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## ROCKET PASTE AREA, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]

CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
24DNT	1.4E+03	8.8E+01	6.9E+01	3.8E+01	1.2E+02
26DNT	5.7E+01	3.5E+00	2.8E+00	1.5E+00	5.0E+00
BAANTR	1.1E+00	5.8E-02	5.6E-02	2.6E-02	8.7E-02
CR	3.8E+01	2.3E+00	1.9E+00	1.1E+00	3.8E+00
DEP	8.4E+01	4.9E+00	4.2E+00	2.2E+00	7.1E+00
HG	5.5E-01	3.9E-02	3.1E-02	6.6E-02	2.3E-01
NG	2.6E+03	1.6E+02	1.3E+02	7.0E+01	2.3E+02
NNDPA	1.7E+04	1.0E+03	8.5E+02	4.4E+02	1.4E+03
PB	1.3E+04	7.2E+02	6.5E+02	2.2E+02	5.6E+02
PYR	1.5E+00	8.3E-02	7.8E-02	3.7E-02	1.2E-01
123PDA	3.3E+01	2.1E+00	1.6E+00	8.9E-01	2.9E+00
CHRY	1.6E+00	8.8E-02	8.4E-02	3.9E-02	1.3E-01
FANT	1.8E+00	9.9E-02	9.4E-02	4.5E-02	1.5E-01
NIT	2.1E+01	1.3E+00	9.5E-01	3.7E-01	1.0E+00
NNDMEA	5.3E-01	3.3E-02	2.6E-02	1.4E-02	4.6E-02
NNDNPA	4.1E-01	2.5E-02	2.0E-02	1.1E-02	3.5E-02
PHANTR	4.5E-01	2.5E-02	2.3E-02	1.1E-02	3.7E-02
SO4	3.9E+00	2.4E-01	1.8E-01	7.0E-02	1.9E-01

TABLE R-24  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
ROCKET PASTE AREA, BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet					Home Range (acres)	Soil	Birds	Herpeto-fauna	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Small	Herpeto-fauna							
<i>Short-tailed shrew</i> (Small Mammal)	85%	10%	0%	0%	0%	1.3	5%	0%	0%	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i> (Small Bird)	75%	20%	0%	0%	0%	5	5%	0%	0%	1.0E+00	0.0095	0.087
<i>Garter snake</i> (Herpetile)	85%	0%	5%	5%	0%	5	5%	5%	0%	1.0E+00	0.023	0.27
<i>Red fox</i> (Pred. Mammal)	20%	10%	40%	40%	15%	250	5%	10%	15%	1.0E+00	0.23	4.9
<i>Red-tailed hawk</i> (Pred. Bird)	5%	5%	55%	55%	10%	500	5%	20%	10%	1.0E+00	0.23	1.5

NOTES:

- [a] Bioaccumulation data presented in: Appendix Q, Table Q-1  
 [b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.  
 [c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2  
 [d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-25  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
NITROGLYCERINE POND, BADGER ARMY AMMUNITION PLANT

EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
HG	2.4E+00
NG	1.6E+01
NH3	1.8E+01
PB	1.0E+04

ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert BAF [a]	Tissue		Plant BAF [a]	Tissue Level (mg/kg)
	Level (mg/kg)	Level (mg/kg)		
3.4E-01	8.2E-01	1.0E+00	2.4E+00	
1.0E+00	1.6E+01	1.0E+00	1.6E+01	
5.0E-02	8.9E-01	5.0E-02	8.9E-01	
2.4E+00	2.4E+04	2.0E-01	2.0E+03	

BAF VALUES FOR OTHER PREY ITEM

Small Mammal BAF	Small		Herptile BAF
	Bird BAF	Bird BAF	
5.0E+00	2.3E+00	1.0E+00	1.0E+01
1.0E+00	1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00	0.0E+00
4.0E-01	3.8E-01	1.0E+00	1.0E+00

SITE AREA: 2.00 acres

TABLE R-25  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
NITROGLYCERINE POND, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]					
CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
HG	1.9E+00	1.3E-01	9.7E-02	1.6E-01	5.9E-01
NG	2.8E+01	1.7E+00	1.3E+00	6.3E-01	2.0E+00
NH3	3.0E+00	1.9E-01	1.4E-01	5.4E-02	1.5E-01
PB	3.8E+04	2.1E+03	1.9E+03	5.0E+02	1.2E+03

TABLE R-25  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
NITROGLYCERINE POND, BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet					Soil	Home Range (acres)	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverte	Plants	Small Mammals	Herpeto-fauna	Birds					
<i>Short-tailed shrew</i> (Small Mammal)	85%	10%	0%	0%	0%	5%	1.3	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i> (Small Bird)	75%	20%	0%	0%	0%	5%	5	4.0E-01	0.0095	0.087
<i>Garter snake</i> (Herptile)	85%	0%	5%	0%	5%	5%	5	4.0E-01	0.023	0.27
<i>Red fox</i> (Pred. Mammal)	20%	10%	40%	15%	10%	5%	250	8.0E-03	0.23	4.9
<i>Red-tailed hawk</i> (Pred. Bird)	5%	5%	55%	10%	20%	5%	500	4.0E-03	0.23	1.5

NOTES:

- [a] Bioaccumulation data presented in: Appendix Q, Table Q-1  
 [b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.  
 [c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2  
 [d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)



TABLE R-26

ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

NITROGLYCERINE POND, BADGER ARMY AMMUNITION PLANT

## EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
HG	2.4E+00
NG	1.6E+01
NH3	1.8E+01
PB	1.0E+04

## ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert BAF [a]	Tissue		Plant BAF [a]	Tissue Level (mg/kg)
	Level (mg/kg)			
3.4E-01	8.2E-01		1.0E+00	2.4E+00
1.0E+00	1.6E+01		1.0E+00	1.6E+01
5.0E-02	8.9E-01		5.0E-02	8.9E-01
2.4E+00	2.4E+04		2.0E-01	2.0E+03

## BAF VALUES FOR OTHER PREY ITEM

Mammal BAF	Small		Herptile BAF
	Bird BAF		
5.0E+00	2.3E+00		1.0E+01
1.0E+00	1.0E+00		1.0E+00
0.0E+00	0.0E+00		0.0E+00
4.0E-01	3.8E-01		1.0E+00

SITE AREA: 2.00 acres

10-Nov-92

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TABLE R-26  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
NITROGLYCERINE POND, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]					
CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
HG	1.9E+00	5.3E-02	3.9E-02	1.3E-03	2.4E-03
NG	2.1E+01	6.9E-01	5.2E-01	5.0E-03	7.9E-03
NH3	3.0E+00	7.5E-02	5.6E-02	4.3E-04	6.0E-04
PB	3.8E+04	8.4E+02	7.4E+02	4.0E+00	4.9E+00

TABLE R-26  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
NITROGLYCERINE POND, BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet				Home Range (acres)	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Herpeto-fauna				
<i>Short-tailed shrew</i> (Small Mammal)	85%	10%	0%	0%	1.3	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i> (Small Bird)	75%	20%	0%	0%	5	4.0E-01	0.0095	0.087
<i>Garter snake</i> (Herpitle)	85%	0%	5%	0%	5	4.0E-01	0.023	0.27
<i>Red fox</i> (Pred. Mammal)	20%	10%	40%	15%	250	8.0E-03	0.23	4.9
<i>Red-tailed hawk</i> (Pred. Bird)	5%	5%	55%	10%	500	4.0E-03	0.23	1.5

NOTES:

- [a] Bioaccumulation data presented in: Appendix Q, Table Q-1  
 [b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.  
 [c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2  
 [d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-27

## ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## OLEUM PLANT POND, BADGER ARMY AMMUNITION PLANT

## EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
SO4	8.5E+03
NIT	3.5E+00

## ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert	BAF [a]	Tissue Level (mg/kg)	Plant BAF [a]	Tissue Level (mg/kg)
5.0E-02	5.0E-02	4.3E+02	5.0E-02	4.3E+02
5.0E-02	5.0E-02	1.7E-01	5.0E-02	1.7E-01

## BAF VALUES FOR OTHER PREY ITEM

Small Mammal BAF	Small Bird BAF	Herpetile BAF
0.0E+00	0.0E+00	0.0E+00
0.0E+00	0.0E+00	0.0E+00

SITE AREA: 4.65 acres

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TABLE R-27  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
OLEUM PLANT POND, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]					
CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
SO4	1.5E+03	9.0E+01	6.7E+01	2.6E+01	7.2E+01
NIT	5.9E-01	3.7E-02	2.7E-02	1.1E-02	2.9E-02

TABLE R-27  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
OLEUM PLANT POND, BADGER ARMY AMMUNITION PLANT

EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet				Soil	Home Range (acres)	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Herpeto-fauna					
<i>Short-tailed shrew</i>	(Small Mammal)	85%	10%	0%	0%	1.3	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i>	(Small Bird)	75%	0%	0%	0%	5	9.3E-01	0.0095	0.087
<i>Garter snake</i>	(Herptile)	85%	0%	0%	5%	5	9.3E-01	0.023	0.27
<i>Red fox</i>	(Pred. Mammal)	20%	40%	15%	10%	250	1.9E-02	0.23	4.9
<i>Red-tailed hawk</i>	(Pred. Bird)	5%	55%	10%	20%	500	9.3E-03	0.23	1.5

NOTES:

- [a] Bioaccumulation data presented in: Appendix Q, Table Q-1  
 [b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.  
 [c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2  
 [d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-28  
 ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
 OLEUM PLANT POND, BADGER ARMY AMMUNITION PLANT

EXPOSURE CONCENTRATION DATA		ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS				BAF VALUES FOR OTHER PREY ITEM			
CHEMICAL	CONCENTRATION (mg/kg)	Invert BAF [a]	Tissue Level (mg/kg)	Plant BAF [a]	Tissue Level (mg/kg)	Small Mammal BAF	Small Bird BAF	Herptile BAF	
SO4	8.5E+03	5.0E-02	4.3E+02	5.0E-02	4.3E+02	0.0E+00	0.0E+00	0.0E+00	
NIT	3.5E+00	5.0E-02	1.7E-01	5.0E-02	1.7E-01	0.0E+00	0.0E+00	0.0E+00	

SITE AREA: 4.65 acres

TABLE R-28  
 ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
 REMEDIAL INVESTIGATION  
 OLEUM PLANT POND, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]					
CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
SO4	1.5E+03	8.4E+01	6.2E+01	4.8E-01	6.7E-01
NIT	5.9E-01	3.4E-02	2.5E-02	2.0E-04	2.7E-04



TABLE R-28

## ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## OLEUM PLANT POND, BADGER ARMY AMMUNITION PLANT

## EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet					Home Range (acres)	Soil	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverte	Plants	Small Mammals	Herpeto-fauna	Birds					
<i>Short-tailed shrew</i> (Small Mammal)	85%	10%	0%	0%	0%	1.3	5%	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i> (Small Bird)	75%	20%	0%	0%	0%	5	5%	9.3E-01	0.0095	0.087
<i>Garter snake</i> (Herptile)	85%	0%	5%	0%	5%	5	5%	9.3E-01	0.023	0.27
<i>Red fox</i> (Pred. Mammal)	20%	10%	40%	15%	10%	250	5%	1.9E-02	0.23	4.9
<i>Red-tailed hawk</i> (Pred. Bird)	5%	5%	55%	10%	20%	500	5%	9.3E-03	0.23	1.5

## NOTES:

[a] Bioaccumulation data presented in: Appendix Q, Table Q-1

[b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.

[c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2

[d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-29

ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

OLD ACID AREA, BADGER ARMY AMMUNITION PLANT

## EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
NI	5.7E+01
NIT	1.8E+00
PB	1.5E+03
SO4	1.8E+04

## ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert BAF [a]	Tissue		Plant BAF [a]	Tissue Level (mg/kg)
	Level (mg/kg)	Level (mg/kg)		
1.9E+00	1.1E+02	1.8E+00	3.2E+00	1.8E+02
5.0E-02	9.0E-02	9.0E-02	5.0E-02	9.0E-02
2.4E+00	3.6E+03	3.0E+02	2.0E-01	3.0E+02
5.0E-02	9.0E+02	9.0E+02	5.0E-02	9.0E+02

## BAF VALUES FOR OTHER PREY ITEM

Mammal BAF	Small		Herptile BAF
	Bird BAF	Bird BAF	
1.2E-01	1.0E+00	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00	0.0E+00
4.3E-01	3.8E-01	3.8E-01	1.0E+00
0.0E+00	0.0E+00	0.0E+00	0.0E+00

SITE AREA: 10.33 acres

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TABLE R-29  
ESTIMATION OF ACUTE EXPOSURES TO TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
OLD ACID AREA, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]					
CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
NI	1.9E+02	1.3E+01	8.4E+00	3.5E+00	8.9E+00
NIT	3.1E-01	1.9E-02	1.4E-02	5.5E-03	1.5E-02
PB	5.6E+03	3.1E+02	2.8E+02	9.2E+01	2.4E+02
SO4	3.1E+03	1.9E+02	1.4E+02	5.5E+01	1.5E+02



TABLE R-30

## ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## OLD ACID AREA, BADGER ARMY AMMUNITION PLANT

## EXPOSURE CONCENTRATION DATA

CHEMICAL	CONCENTRATION (mg/kg)
SO4	1.8E+04
PB	1.5E+03
NI	5.7E+01
NIT	1.8E+00

## ESTIMATED TISSUE LEVELS IN PRIMARY PREY ITEMS

Invert BAF [a]	Tissue Level (mg/kg)	Plant BAF [a]	Tissue Level (mg/kg)
5.0E-02	9.0E+02	5.0E-02	9.0E+02
2.4E+00	3.6E+03	2.0E-01	3.0E+02
1.9E+00	1.1E+02	3.2E+00	1.8E+02
5.0E-02	9.0E-02	5.0E-02	9.0E-02

## BAF VALUES FOR OTHER PREY ITEM

Small Mammal BAF	Small Bird BAF	Herpille BAF
0.0E+00	0.0E+00	0.0E+00
4.3E-01	3.8E-01	1.0E+00
1.2E-01	1.0E+00	1.0E+00
0.0E+00	0.0E+00	0.0E+00

SITE AREA: 10.33 acres

10-Nov-92

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TABLE R-30  
ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
OLD ACID AREA, BADGER ARMY AMMUNITION PLANT

TOTAL BODY DOSE (mg/kgBW-day) [b]					
CHEMICAL	Short-tailed shrew	Eastern meadowlark	Garter snake	Red fox	Red-tailed hawk
SO4	3.1E+03	1.9E+02	1.4E+02	2.3E+00	3.1E+00
PB	5.6E+03	3.1E+02	2.8E+02	3.9E+00	5.0E+00
NI	1.9E+02	1.3E+01	8.4E+00	1.4E-01	1.8E-01
NIT	3.1E-01	1.9E-02	1.4E-02	2.3E-04	3.1E-04

TABLE R-30

## ESTIMATION OF CHRONIC EXPOSURES TO INDIVIDUAL TERRESTRIAL ORGANISMS VIA FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## OLD ACID AREA, BADGER ARMY AMMUNITION PLANT

## EXPOSURE PARAMETERS [c]

Indicator Species	Percent Prey in Diet					Soil	Home Range (acres)	Site Foraging Frequency [d]	Ingestion Rate (kg/day)	Body Weight (kg)
	Inverts	Plants	Small Mammals	Herpeto-fauna	Birds					
<i>Short-tailed shrew</i> (Small Mammal)	85%	10%	0%	0%	0%	5%	1.3	1.0E+00	0.037	0.021
<i>Eastern meadowlark</i> (Small Bird)	75%	20%	0%	0%	0%	5%	5	1.0E+00	0.0095	0.087
<i>Garter snake</i> (Herpetile)	85%	0%	5%	0%	5%	5%	5	1.0E+00	0.023	0.27
<i>Red fox</i> (Pred. Mammal)	20%	10%	40%	15%	10%	5%	250	4.1E-02	0.23	4.9
<i>Red-tailed hawk</i> (Pred. Bird)	5%	5%	55%	10%	20%	5%	500	2.1E-02	0.23	1.5

## NOTES:

[a] Bioaccumulation data presented in: Appendix I, Table Q-1

[b] Calculated by summing the products of individual prey type concentrations and percent in diet, multiplying by the SFF and ingestion rate, and then dividing by body weight.

[c] Documentation of exposure parameters presented in: Appendix Q, Table Q-2

[d] Site Foraging Frequency (SFF). Calculated by dividing site area by receptor home range (cannot exceed 1.0)

TABLE R-31

ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

PROPELLANT BURNING GROUND, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Short-tailed shrew			Eastern meadowlark			Garter snake		
	TBD	RTV	HI	TBD	RTV	HI	TBD	RTV	HI
24DNT	1.9E+01	5.4E+01	3.5E-01	1.2E+00	5.4E+01	2.2E-02	9.1E-01	5.4E+01	1.7E-02
2MNAP	7.2E-01	3.3E+02	2.2E-03	3.9E-02	3.3E+02	1.2E-04	3.8E-02	3.3E+02	1.1E-04
AS	1.5E+01	7.5E+01	2.0E-01	8.7E-01	9.5E+00	9.1E-02	7.6E-01	9.5E+00	8.0E-02
BAANTR	3.2E-01	2.0E+01	1.6E-02	1.8E-02	2.0E+01	9.0E-04	1.7E-02	2.0E+01	8.6E-04
B2EHP	9.9E+00	1.7E+03	5.7E-03	5.5E-01	1.7E+03	3.2E-04	5.2E-01	1.7E+03	3.0E-04
C6H6	7.4E-01	1.0E+02	7.4E-03	4.6E-02	1.0E+02	4.6E-04	3.6E-02	1.0E+02	3.6E-04
CHRY	5.8E+00	9.9E+02	5.9E-03	3.2E-01	9.9E+02	3.3E-04	3.1E-01	9.9E+02	3.1E-04
CR	1.7E+01	6.0E+01	2.9E-01	1.0E+00	2.5E+01	4.1E-02	8.7E-01	2.5E+01	3.5E-02
CU	5.4E+03	1.2E+01	4.5E+02	3.4E+02	2.1E+00	1.6E+02	2.6E+02	2.1E+00	1.2E+02
DEP	1.0E+01	1.7E+03	6.1E-03	6.1E-01	1.7E+03	3.6E-04	5.2E-01	1.7E+03	3.0E-04
DNBP	1.0E+01	6.0E+03	1.7E-03	5.6E-01	6.0E+03	9.4E-05	5.3E-01	6.0E+03	8.9E-05
FANT	3.2E-01	4.0E+02	8.0E-04	1.8E-02	4.0E+02	4.4E-05	1.7E-02	4.0E+02	4.2E-05
HG	2.6E-01	3.6E+00	7.2E-02	1.8E-02	4.0E-01	4.6E-02	1.4E-02	4.0E-01	3.6E-02
NI	9.5E+01	1.3E+01	7.1E+00	6.3E+00	1.0E+02	6.3E-02	4.1E+00	1.3E+01	3.1E-01
NNDPA	5.2E+01	5.0E+02	1.0E-01	3.1E+00	5.0E+02	6.2E-03	2.6E+00	5.0E+02	5.2E-03
PB	1.0E+04	2.0E+00	5.0E+03	5.6E+02	4.9E+00	1.1E+02	5.0E+02	2.0E+00	2.5E+02
PHANTR	2.1E+00	1.4E+02	1.5E-02	1.2E-01	1.4E+02	8.4E-04	1.1E-01	1.4E+02	7.9E-04
PYR	2.7E-01	1.6E+02	1.7E-03	1.5E-02	1.6E+02	9.3E-05	1.4E-02	1.6E+02	8.8E-05
SE	1.1E+00	4.0E-02	2.7E+01	6.7E-02	6.0E-01	1.1E-01	5.3E-02	4.0E-02	1.3E+00
ZN	1.3E+04	5.0E+02	2.7E+01	8.5E+02	5.0E+02	1.7E+00	1.1E+03	5.0E+02	2.1E+00
SUMMARY HAZARD INDEX						2.8E+02			3.8E+02



TABLE R-31  
ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
PROPELLANT BURNING GROUND, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Red fox			Red-tailed hawk		
	TBD	RTV	HI	TBD	RTV	HI
24DNT	5.0E-01	5.0E+00	1.0E-01	1.6E+00	5.4E+01	3.0E-02
2MNAP	1.8E-02	3.3E+02	5.4E-05	5.9E-02	3.3E+02	1.8E-04
AS	2.6E-01	2.5E+03	1.1E-04	7.0E-01	9.5E+00	7.4E-02
BAANTR	8.1E-03	2.0E+01	4.0E-04	2.7E-02	2.0E+01	1.3E-03
B2EHP	2.5E-01	1.7E+03	1.4E-04	8.2E-01	1.7E+03	4.8E-04
C6H6	2.0E-02	1.0E+02	2.0E-04	6.4E-02	1.0E+02	6.4E-04
CHRY	1.5E-01	9.9E+02	1.5E-04	4.8E-01	9.9E+02	4.9E-04
CR	5.1E-01	6.0E+01	8.6E-03	1.8E+00	2.5E+01	7.0E-02
CU	1.4E-02	1.2E+01	1.2E-01	4.6E+02	2.1E+00	2.2E-02
DEP	2.7E-01	1.7E+03	1.6E-04	8.9E-01	1.7E+03	5.1E-04
DNBP	2.5E-01	6.0E+03	4.2E-05	8.4E-01	6.0E+03	1.4E-04
FANT	8.0E-03	4.0E+02	2.0E-05	2.6E-02	4.0E+02	6.6E-05
HG	3.1E-02	1.0E+00	3.1E-02	1.1E-01	4.0E-01	2.6E-01
NI	1.7E+00	6.3E+02	2.7E-03	4.3E+00	1.0E+02	4.3E-02
NNDPA	1.4E+00	5.0E+02	2.7E-03	4.4E+00	5.0E+02	8.9E-03
PB	1.7E+02	3.0E+01	5.5E+00	4.3E+02	2.5E+01	1.7E+01
PHANTR	5.3E-02	1.4E+02	3.8E-04	1.8E-01	1.4E+02	1.3E-03
PYR	6.7E-03	1.6E+02	4.2E-05	2.2E-02	1.6E+02	1.4E-04
SE	2.9E-02	4.0E-02	7.3E-01	9.5E-02	6.0E-01	1.6E-01
ZN	2.1E+03	5.0E+02	4.2E+00	7.7E+03	5.0E+02	1.5E+01
SUMMARY HAZARD INDEX			2.2E+01	2.5E+02		

NOTES: TBD = Total Body Dose (mg/lr) ; W-day) BW = Body Weight (kg)  
RTV = Reference Toxicity Vr (mg/kgBW-day) HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-32

ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

PROPELLANT BURNING GROUND, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Short-tailed shrew			Eastern meadowlark			Garter snake		
	TBD	RTV	HI	TBD	RTV	HI	TBD	RTV	HI
24DNT	1.9E+01	4.0E+01	4.7E-01	1.2E+00	4.0E+01	2.9E-02	9.1E-01	4.0E+01	2.3E-02
2MNAP	7.2E-01	3.3E+01	2.2E-02	3.9E-02	3.3E+01	1.2E-03	3.8E-02	3.3E+01	1.1E-03
AS	1.5E+01	7.5E+00	2.0E+00	8.7E-01	1.0E+00	8.7E-01	7.6E-01	1.0E+00	7.6E-01
BAANTR	3.2E-01	2.0E+00	1.6E-01	1.8E-02	2.0E+00	9.0E-03	1.7E-02	2.0E+00	8.6E-03
B2EHP	9.9E+00	1.9E+01	5.2E-01	5.5E-01	1.9E+01	2.9E-02	5.2E-01	1.9E+01	2.7E-02
C6H6	7.4E-01	1.0E+01	7.4E-02	4.6E-02	1.0E+01	4.6E-03	3.6E-02	1.0E+01	3.6E-03
CHRY	5.8E+00	9.9E+01	5.9E-02	3.2E-01	9.9E+01	3.3E-03	3.1E-01	9.9E+01	3.1E-03
CR	1.7E+01	5.7E+00	3.0E+00	1.0E+00	3.5E+00	3.0E-01	8.7E-01	3.5E+00	2.5E-01
CU	5.4E+03	1.2E+00	4.5E+03	3.4E+02	2.0E-01	1.7E+03	2.6E+02	2.0E-01	1.3E+03
DEP	1.0E+01	1.7E+02	6.1E-02	6.1E-01	1.7E+02	3.6E-03	5.2E-01	1.7E+02	3.0E-03
DNBP	1.0E+01	6.0E+02	1.7E-02	5.6E-01	6.0E+02	9.4E-04	5.3E-01	6.0E+02	8.9E-04
FANT	3.2E-01	4.0E+01	8.0E-03	1.8E-02	4.0E+01	4.4E-04	1.7E-02	4.0E+01	4.2E-04
HG	2.6E-01	1.2E-01	2.2E+00	1.8E-02	7.0E-03	2.6E+00	1.4E-02	7.0E-03	2.1E+00
NI	9.5E+01	1.3E+00	7.3E+01	6.3E+00	1.0E+01	6.2E-01	4.1E+00	1.3E+00	3.2E+00
NNDPA	5.2E+01	5.0E+01	1.0E+00	3.1E+00	5.0E+01	6.2E-02	2.6E+00	5.0E+01	5.2E-02
PB	1.0E+04	1.0E-01	1.0E+05	5.6E+02	1.8E+00	3.2E+02	5.0E+02	1.0E-01	5.0E+03
PHANTR	2.1E+00	1.4E+01	1.5E-01	1.2E-01	1.4E+01	8.4E-03	1.1E-01	1.4E+01	7.9E-03
PYR	2.7E-01	1.3E+02	2.1E-03	1.5E-02	1.3E+02	1.2E-04	1.4E-02	1.3E+02	1.1E-04
SE	1.1E+00	4.0E-03	2.7E+02	6.7E-02	6.0E-02	1.1E+00	5.3E-02	4.0E-03	1.3E+01
ZN	1.3E+04	1.6E+02	8.3E+01	8.5E+02	1.6E+02	5.3E+00	1.1E+03	1.6E+02	6.6E+00
SUMMARY HAZARD INDEX			1.1E+05			2.0E+03			6.3E+03

TABLE R-32  
ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
PROPELLANT BURNING GROUND, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Red fox			Red-tailed hawk		
	TBD	RTV	HI	TBD	RTV	HI
24DNT	9.6E-02	1.0E+00	9.6E-02	1.6E-01	4.0E+01	3.9E-03
2MNAP	3.4E-03	3.3E+01	1.0E-04	5.7E-03	3.3E+01	1.7E-04
AS	5.1E-02	2.5E+02	2.0E-04	6.8E-02	1.0E+00	6.8E-02
BAANTR	1.5E-03	2.0E+00	7.7E-04	2.6E-03	2.0E+00	1.3E-03
B2EHP	4.7E-02	1.9E+01	2.5E-03	7.8E-02	1.9E+01	4.1E-03
C6H6	3.8E-03	1.0E+01	3.8E-04	6.2E-03	1.0E+01	6.2E-04
CHRY	2.8E-02	9.9E+01	2.8E-04	4.6E-02	9.9E+01	4.7E-04
CR	9.9E-02	5.7E+00	1.7E-02	1.7E-01	3.5E+00	4.8E-02
CU	2.7E+01	1.2E+00	2.2E+01	4.4E+01	2.0E-01	2.2E+02
DEP	5.2E-02	1.7E+02	3.0E-04	8.5E-02	1.7E+02	4.9E-04
DNBP	4.9E-02	6.0E+02	8.1E-05	8.1E-02	6.0E+02	1.3E-04
FANT	1.5E-03	4.0E+01	3.8E-05	2.5E-03	4.0E+01	6.3E-05
HG	5.9E-03	1.0E-01	5.9E-02	1.0E-02	7.0E-03	1.4E+00
NI	3.3E-01	6.3E+01	5.2E-03	4.2E-01	1.0E+01	4.1E-02
NNDPA	2.6E-01	5.0E+01	5.2E-03	4.3E-01	5.0E+01	8.5E-03
PB	3.2E+01	3.0E+00	1.1E+01	4.1E+01	2.5E+00	1.7E+01
PHANTR	1.0E-02	1.4E+01	7.3E-04	1.7E-02	1.4E+01	1.2E-03
PYR	1.3E-03	1.3E+02	1.0E-05	2.1E-03	1.3E+02	1.7E-05
SE	5.6E-03	4.0E-03	1.4E+00	9.1E-03	6.0E-02	1.5E-01
ZN	4.0E+02	1.6E+02	2.5E+00	7.4E+02	1.6E+02	4.6E+00
SUMMARY HAZARD INDEX			3.7E+01			2.4E+02

NOTES: TBD = Total Body Dose (mg/kgBW-day)      BW = Body Weight (kg)  
RTV = Reference Toxicity Value (mg/kgBW-day)      HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-33

## ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## FINAL CREEK, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Short-tailed shrew</i>				<i>Eastern meadowlark</i>				<i>Garter snake</i>			
	TBD	RTV	HI	HI	TBD	RTV	HI	HI	TBD	RTV	HI	HI
SO4	4.5E+01	1.2E+03	3.7E-02	3.7E-02	2.8E+00	1.2E+03	2.3E-03	2.3E-03	2.0E+00	1.2E+03	1.7E-03	1.7E-03
PB	1.5E+02	2.0E+00	7.5E+01	7.5E+01	8.4E+00	4.9E+00	1.7E+00	1.7E+00	7.4E+00	2.0E+00	3.7E+00	3.7E+00
24DNT	1.1E+01	5.4E+01	2.0E-01	2.0E-01	6.6E-01	5.4E+01	1.2E-02	1.2E-02	5.0E-01	5.4E+01	9.2E-03	9.2E-03
26DNT	7.0E+01	5.4E+01	1.3E+00	1.3E+00	4.4E+00	5.4E+01	8.1E-02	8.1E-02	3.3E+00	5.4E+01	6.1E-02	6.1E-02
NIT	1.9E+00	1.3E+03	1.4E-03	1.4E-03	1.2E-01	1.3E+03	8.8E-05	8.8E-05	8.7E-02	1.3E+03	6.5E-05	6.5E-05
SN	1.1E+02	3.8E+01	3.0E+00	3.0E+00	6.9E+00	3.5E+01	2.0E-01	2.0E-01	5.2E+00	3.5E+01	1.5E-01	1.5E-01
DEP	2.2E-01	1.7E+03	1.3E-04	1.3E-04	1.3E-02	1.7E+03	7.5E-06	7.5E-06	1.1E-02	1.7E+03	6.2E-06	6.2E-06
DNBP	4.2E+01	6.0E+03	6.9E-03	6.9E-03	2.3E+00	6.0E+03	3.8E-04	3.8E-04	2.1E+00	6.0E+03	3.5E-04	3.5E-04
DPA	2.6E+01	3.1E+02	8.5E-02	8.5E-02	1.6E+00	3.1E+02	5.3E-03	5.3E-03	1.2E+00	3.1E+02	4.0E-03	4.0E-03
2NDPA	3.4E+00	5.0E+02	6.8E-03	6.8E-03	2.0E-01	5.0E+02	4.0E-04	4.0E-04	1.6E-01	5.0E+02	3.3E-04	3.3E-04
NC	1.3E+02	9.0E+04	1.4E-03	1.4E-03	7.9E+00	9.0E+04	8.8E-05	8.8E-05	5.8E+00	9.0E+04	6.5E-05	6.5E-05
NH3	3.1E+02	3.2E+03	9.7E-02	9.7E-02	1.9E+01	3.2E+03	6.0E-03	6.0E-03	1.4E+01	3.2E+03	4.5E-03	4.5E-03
SUMMARY HAZARD INDEX											3.9E+00	3.9E+00

TABLE R-33  
ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
FINAL CREEK, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Red fox			Red-tailed hawk		
	TBD	RTV	HI	TBD	RTV	HI
SO4	7.9E-01	1.2E+03	6.6E-04	2.2E+00	1.2E+03	1.8E-03
PB	2.0E+00	3.0E+01	6.8E-02	5.1E+00	2.5E+01	2.0E-01
24DNT	2.4E-01	5.0E+00	4.8E-02	7.5E-01	5.4E+01	1.4E-02
26DNT	1.6E+00	5.0E+00	3.2E-01	5.0E+00	5.4E+01	9.3E-02
NIT	3.4E-02	1.3E+03	2.5E-05	9.3E-02	1.3E+03	7.0E-05
SN	2.5E+00	3.8E+01	6.7E-02	7.9E+00	3.5E+01	2.3E-01
DEP	4.8E-03	1.7E+03	2.8E-06	1.5E-02	1.7E+03	8.8E-06
DNBP	8.7E-01	6.0E+03	1.4E-04	2.8E+00	6.0E+03	4.7E-04
DPA	6.0E-01	2.5E+02	2.4E-03	1.9E+00	3.1E+02	6.1E-03
2NDPA	7.4E-02	5.0E+02	1.5E-04	2.4E-01	5.0E+02	4.7E-04
NC	2.3E+00	9.0E+04	2.5E-05	6.2E+00	9.0E+04	6.9E-05
NH3	5.5E+00	3.2E+03	1.7E-03	1.5E+01	3.2E+03	4.8E-03
SUMMARY HAZARD INDEX			5.1E-01	5.5E-01		

NOTES: TBD = Total Body Dose (mg/kgBW-day)      BW = Body Weight (kg)  
RTV = Reference Toxicity Value (mg/kgBW-day)      HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-34  
ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
FINAL CREEK, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Short-tailed shrew</i>				<i>Eastern meadowlark</i>				<i>Garter snake</i>			
	TBD	RTV	HI		TBD	RTV	HI		TBD	RTV	HI	
SO4	4.5E+01	1.2E+02	3.7E-01		1.1E+00	1.2E+02	9.2E-03		8.2E-01	1.2E+02	6.8E-03	
PB	1.5E+02	1.0E-01	1.5E+03		3.3E+00	1.8E+00	1.9E+00		3.0E+00	1.0E-01	3.0E+01	
24DNT	1.1E+01	4.0E+01	2.6E-01		2.6E-01	4.0E+01	6.6E-03		2.0E-01	4.0E+01	5.0E-03	
26DNT	7.0E+01	4.0E+01	1.8E+00		1.7E+00	4.0E+01	4.4E-02		1.3E+00	4.0E+01	3.3E-02	
NIT	1.9E+00	1.3E+02	1.4E-02		4.7E-02	1.3E+02	3.5E-04		3.5E-02	1.3E+02	2.6E-04	
SN	1.1E+02	1.0E-01	1.1E+03		2.8E+00	3.5E+00	7.9E-01		2.1E+00	1.0E-01	2.1E+01	
DEP	2.2E-01	1.7E+02	1.3E-03		5.1E-03	1.7E+02	3.0E-05		4.3E-03	1.7E+02	2.5E-05	
DNBP	4.2E+01	6.0E+02	6.9E-02		9.2E-01	6.0E+02	1.5E-03		8.5E-01	6.0E+02	1.4E-03	
DPA	2.6E+01	3.1E+01	8.5E-01		6.6E-01	3.1E+01	2.1E-02		5.0E-01	3.1E+01	1.6E-02	
2NDPA	3.4E+00	5.0E+01	6.8E-02		8.0E-02	5.0E+01	1.6E-03		6.6E-02	5.0E+01	1.3E-03	
NC	1.3E+02	9.0E+03	1.4E-02		3.2E+00	9.0E+03	3.5E-04		2.3E+00	9.0E+03	2.6E-04	
NH3	3.1E+02	9.4E+02	3.3E-01		7.7E+00	9.4E+02	8.2E-03		5.7E+00	9.4E+02	6.1E-03	
SUMMARY HAZARD INDEX				2.6E+03	2.8E+00				5.1E+01			

TABLE R-34  
ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
FINAL CREEK, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Red fox</i>			<i>Red-tailed hawk</i>		
	TBD	RTV	HI	TBD	RTV	HI
SO4	6.3E-03	1.2E+02	5.3E-05	8.8E-03	1.2E+02	7.3E-05
PB	1.6E-02	3.0E+00	5.4E-03	2.0E-02	2.5E+00	8.2E-03
24DNT	1.9E-03	1.0E+00	1.9E-03	3.0E-03	4.0E+01	7.5E-05
26DNT	1.3E-02	1.0E+00	1.3E-02	2.0E-02	4.0E+01	5.0E-04
NIT	2.7E-04	1.3E+02	2.0E-06	3.7E-04	1.3E+02	2.8E-06
SN	2.0E-02	1.0E-01	2.0E-01	3.2E-02	3.5E+00	9.0E-03
DEP	3.8E-05	1.7E+02	2.2E-07	6.1E-05	1.7E+02	3.5E-07
DNBP	6.9E-03	6.0E+02	1.2E-05	1.1E-02	6.0E+02	1.9E-05
DPA	4.8E-03	2.5E+01	1.9E-04	7.5E-03	3.1E+01	2.4E-04
2NDPA	5.9E-04	5.0E+01	1.2E-05	9.4E-04	5.0E+01	1.9E-05
NC	1.8E-02	9.0E+03	2.0E-06	2.5E-02	9.0E+03	2.8E-06
NH3	4.4E-02	3.2E+02	1.4E-04	6.1E-02	9.4E+02	6.5E-05
SUMMARY HAZARD INDEX			2.2E-01	1.8E-02		

NOTES: TBD = Total Body Dose (mg/kgBW-day)      BW = Body Weight (kg)  
RTV = Reference Toxicity Value (mg/kgBW-day)      HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-35

## ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

 REMEDIAL INVESTIGATION  
 SETTLING POND 1, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Short-tailed shrew			Eastern meadowlark			Garter snake		
	TBD	RTV	HI	TBD	RTV	HI	TBD	RTV	HI
SO4	4.3E+02	1.2E+03	3.6E-01	2.7E+01	1.2E+03	2.2E-02	2.0E+01	1.2E+03	1.6E-02
PB	6.8E+02	2.0E+00	3.4E+02	3.8E+01	4.9E+00	7.7E+00	3.4E+01	2.0E+00	1.7E+01
24DNT	3.0E+02	5.4E+01	5.6E+00	1.9E+01	5.4E+01	3.5E-01	1.5E+01	5.4E+01	2.7E-01
26DNT	4.6E+01	5.4E+01	8.5E-01	2.8E+00	5.4E+01	5.3E-02	2.2E+00	5.4E+01	4.1E-02
NIT	2.2E+00	1.3E+03	1.7E-03	1.4E-01	1.3E+03	1.0E-04	1.0E-01	1.3E+03	7.7E-05
SN	1.0E+02	3.8E+01	2.7E+00	6.2E+00	3.5E+01	1.8E-01	4.9E+00	3.5E+01	1.4E-01
DEP	7.7E+02	1.7E+03	4.5E-01	4.6E+01	1.7E+03	2.6E-02	3.9E+01	1.7E+03	2.3E-02
DNBP	2.2E+01	6.0E+03	3.7E-03	1.2E+00	6.0E+03	2.1E-04	1.2E+00	6.0E+03	2.0E-04
DPA	1.8E+01	3.1E+02	5.7E-02	1.1E+00	3.1E+02	3.5E-03	8.5E-01	3.1E+02	2.7E-03
2NDPA	1.6E+00	5.0E+02	3.3E-03	9.7E-02	5.0E+02	1.9E-04	8.2E-02	5.0E+02	1.6E-04
NC	1.0E+04	9.0E+04	1.1E-01	6.4E+02	9.0E+04	7.1E-03	4.7E+02	9.0E+04	5.3E-03
NH3	1.3E+02	3.2E+03	4.0E-02	7.9E+00	3.2E+03	2.5E-03	5.8E+00	3.2E+03	1.8E-03
SUMMARY HAZARD INDEX			3.5E+02			8.3E+00			1.7E+01



TABLE R-35  
ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SETTLING POND 1, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Red fox</i>			<i>Red-tailed hawk</i>		
	TBD	RTV	HI	TBD	RTV	HI
SO4	7.6E+00	1.2E+03	6.4E-03	2.1E+01	1.2E+03	1.8E-02
PB	1.1E+01	3.0E+01	3.7E-01	2.9E+01	2.5E+01	1.2E+00
24DNT	8.1E+00	5.0E+00	1.6E+00	2.6E+01	5.4E+01	4.9E-01
26DNT	1.2E+00	5.0E+00	2.4E-01	4.0E+00	5.4E+01	7.4E-02
NIT	4.0E-02	1.3E+03	3.0E-05	1.1E-01	1.3E+03	8.2E-05
SN	2.7E+00	3.8E+01	7.1E-02	8.7E+00	3.5E+01	2.5E-01
DEP	2.0E+01	1.7E+03	1.2E-02	6.6E+01	1.7E+03	3.8E-02
DNBP	5.6E-01	6.0E+03	9.3E-05	1.9E+00	6.0E+03	3.1E-04
DPA	4.7E-01	2.5E+02	1.9E-03	1.5E+00	3.1E+02	4.9E-03
2NDPA	4.3E-02	5.0E+02	8.5E-05	1.4E-01	5.0E+02	2.8E-04
NC	1.8E+02	9.0E+04	2.0E-03	5.1E+02	9.0E+04	5.6E-03
NH3	2.3E+00	3.2E+03	7.1E-04	6.2E+00	3.2E+03	2.0E-03
SUMMARY HAZARD INDEX			2.3E+00			2.0E+00

NOTES: TBD = Total Body Dose (mg/kgBW-day)      BW = Body Weight (kg)  
RTV = Reference Toxicity Value (mg/kgBW-day)      HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-36

## ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SETTLING POND 1, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Short-tailed shrew</i>				<i>Eastern meadowlark</i>				<i>Garter snake</i>			
	TBD	RTV	HI		TBD	RTV	HI		TBD	RTV	HI	
SO4	4.3E+02	1.2E+02	3.6E+00		2.7E+01	1.2E+02	2.2E-01		2.0E+01	1.2E+02	1.6E-01	
PB	6.8E+02	1.0E-01	6.8E+03		3.8E+01	1.8E+00	2.1E+01		3.4E+01	1.0E-01	3.4E+02	
24DNT	3.0E+02	4.0E+01	7.6E+00		1.9E+01	4.0E+01	4.7E-01		1.5E+01	4.0E+01	3.7E-01	
26DNT	4.6E+01	4.0E+01	1.1E+00		2.8E+00	4.0E+01	7.1E-02		2.2E+00	4.0E+01	5.5E-02	
NIT	2.2E+00	1.3E+02	1.7E-02		1.4E-01	1.3E+02	1.0E-03		1.0E-01	1.3E+02	7.7E-04	
SN	1.0E+02	1.0E-01	1.0E+03		6.2E+00	3.5E+00	1.8E+00		4.9E+00	1.0E-01	4.9E+01	
DEP	7.7E+02	1.7E+02	4.5E+00		4.6E+01	1.7E+02	2.6E-01		3.9E+01	1.7E+02	2.3E-01	
DNBP	2.2E+01	6.0E+02	3.7E-02		1.2E+00	6.0E+02	2.1E-03		1.2E+00	6.0E+02	2.0E-03	
DPA	1.8E+01	3.1E+01	5.7E-01		1.1E+00	3.1E+01	3.5E-02		8.5E-01	3.1E+01	2.7E-02	
2NDPA	1.6E+00	5.0E+01	3.3E-02		9.7E-02	5.0E+01	1.9E-03		8.2E-02	5.0E+01	1.6E-03	
NC	1.0E+04	9.0E+03	1.1E+00		6.4E+02	9.0E+03	7.1E-02		4.7E+02	9.0E+03	5.3E-02	
NH3	1.3E+02	9.4E+02	1.4E-01		7.9E+00	9.4E+02	8.4E-03		5.8E+00	9.4E+02	6.2E-03	
SUMMARY HAZARD INDEX				7.8E+03			2.4E+01				3.9E+02	

TABLE R-36  
ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SETTLING POND 1, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Red fox			Red-tailed hawk		
	TBD	RTV	HI	TBD	RTV	HI
SO4	7.3E-01	1.2E+02	6.1E-03	1.0E+00	1.2E+02	8.4E-03
PB	1.1E+00	3.0E+00	3.6E-01	1.4E+00	2.5E+00	5.6E-01
24DNT	7.8E-01	1.0E+00	7.8E-01	1.3E+00	4.0E+01	3.2E-02
26DNT	1.2E-01	1.0E+00	1.2E-01	1.9E-01	4.0E+01	4.8E-03
NIT	3.8E-03	1.3E+02	2.9E-05	5.3E-03	1.3E+02	4.0E-05
SN	2.6E-01	1.0E-01	2.6E+00	4.2E-01	3.5E+00	1.2E-01
DEP	1.9E+00	1.7E+02	1.1E-02	3.2E+00	1.7E+02	1.8E-02
DNBP	5.4E-02	6.0E+02	8.9E-05	8.9E-02	6.0E+02	1.5E-04
DPA	4.5E-02	2.5E+01	1.8E-03	7.4E-02	3.1E+01	2.4E-03
2NDPA	4.1E-03	5.0E+01	8.2E-05	6.7E-03	5.0E+01	1.3E-04
NC	1.8E+01	9.0E+03	2.0E-03	2.4E+01	9.0E+03	2.7E-03
NH3	2.2E-01	3.2E+02	6.8E-04	3.0E-01	9.4E+02	3.2E-04
SUMMARY HAZARD INDEX			3.8E+00	7.5E-01		

NOTES: TBD = Total Body Dose (mg/kgBW-day)      BW = Body Weight (kg)  
RTV = Reference Toxicity Value (mg/kgBW-day)      HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-37

## ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SETTLING POND 2, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Short-tailed shrew</i>				<i>Eastern meadowlark</i>				<i>Garter snake</i>			
	TBD	RTV	HI		TBD	RTV	HI		TBD	RTV	HI	
SO4	1.1E+01	1.2E+03	9.2E-03		6.8E-01	1.2E+03	5.7E-04		5.0E-01	1.2E+03	4.2E-04	
PB	9.4E+02	2.0E+00	4.7E+02		5.2E+01	4.9E+00	1.1E+01		4.7E+01	2.0E+00	2.3E+01	
24DNT	1.3E+01	5.4E+01	2.5E-01		8.3E-01	5.4E+01	1.5E-02		6.5E-01	5.4E+01	1.2E-02	
NIT	7.4E+00	1.3E+03	5.6E-03		4.6E-01	1.3E+03	3.4E-04		3.4E-01	1.3E+03	2.5E-04	
SN	9.3E+01	3.8E+01	2.5E+00		5.8E+00	3.5E+01	1.7E-01		4.5E+00	3.5E+01	1.3E-01	
DEP	2.3E+02	1.7E+03	1.3E-01		1.3E+01	1.7E+03	7.8E-03		1.1E+01	1.7E+03	6.6E-03	
DNBP	1.2E+00	6.0E+03	2.0E-04		6.6E-02	6.0E+03	1.1E-05		6.2E-02	6.0E+03	1.0E-05	
DPA	2.6E+00	3.1E+02	8.5E-03		1.6E-01	3.1E+02	5.3E-04		1.3E-01	3.1E+02	4.1E-04	
NC	4.8E+01	9.0E+04	5.3E-04		3.0E+00	9.0E+04	3.3E-05		2.2E+00	9.0E+04	2.5E-05	
NH3	1.4E+02	3.2E+03	4.5E-02		8.9E+00	3.2E+03	2.8E-03		6.6E+00	3.2E+03	2.1E-03	
SUMMARY HAZARD INDEX				4.7E+02	1.1E+01				2.4E+01			

TABLE R-37  
ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SETTLING POND 2, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Red fox</i>			<i>Red-tailed hawk</i>		
	TBD	RTV	HI	TBD	RTV	HI
SO4	2.0E-01	1.2E+03	1.6E-04	5.4E-01	1.2E+03	4.5E-04
PB	1.6E+01	3.0E+01	5.2E-01	4.0E+01	2.5E+01	1.6E+00
24DNT	3.6E-01	5.0E+00	7.1E-02	1.2E+00	5.4E+01	2.2E-02
NIT	1.3E-01	1.3E+03	9.9E-05	3.6E-01	1.3E+03	2.7E-04
SN	2.5E+00	3.8E+01	6.6E-02	8.1E+00	3.5E+01	2.3E-01
DEP	5.9E+00	1.7E+03	3.4E-03	1.9E+01	1.7E+03	1.1E-02
DNBP	2.9E-02	6.0E+03	4.9E-06	9.8E-02	6.0E+03	1.6E-05
DPA	7.0E-02	2.5E+02	2.8E-04	2.3E-01	3.1E+02	7.4E-04
NC	8.5E-01	9.0E+04	9.5E-06	2.4E+00	9.0E+04	2.6E-05
NH3	2.6E+00	3.2E+03	8.1E-04	7.1E+00	3.2E+03	2.2E-03
SUMMARY HAZARD INDEX			6.6E-01	1.9E+00		

NOTES: TBD = Total Body Dose (mg/kgBW-day)      BW = Body Weight (kg)  
RTV = Reference Toxicity Value (mg/kgBW-day)      HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-38

## ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SETTLING POND 2, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Short-tailed shrew				Eastern meadowlark				Garter snake			
	TBD	RTV	HI		TBD	RTV	HI		TBD	RTV	HI	
SO4	1.1E+01	1.2E+02	9.2E-02		6.8E-01	1.2E+02	5.7E-03		5.0E-01	1.2E+02	4.2E-03	
PB	9.4E+02	1.0E-01	9.4E+03		5.2E+01	1.8E+00	3.0E+01		4.7E+01	1.0E-01	4.7E+02	
24DNT	1.3E+01	4.0E+01	3.3E-01		8.3E-01	4.0E+01	2.1E-02		6.5E-01	4.0E+01	1.6E-02	
NIT	7.4E+00	1.3E+02	5.6E-02		4.6E-01	1.3E+02	3.4E-03		3.4E-01	1.3E+02	2.5E-03	
SN	9.3E+01	1.0E-01	9.3E+02		5.8E+00	3.5E+00	1.7E+00		4.5E+00	1.0E-01	4.5E+01	
DEP	2.3E+02	1.7E+02	1.3E+00		1.3E+01	1.7E+02	7.8E-02		1.1E+01	1.7E+02	6.6E-02	
DNBP	1.2E+00	6.0E+02	2.0E-03		6.6E-02	6.0E+02	1.1E-04		6.2E-02	6.0E+02	1.0E-04	
DPA	2.6E+00	3.1E+01	8.5E-02		1.6E-01	3.1E+01	5.3E-03		1.3E-01	3.1E+01	4.1E-03	
NC	4.8E+01	9.0E+03	5.3E-03		3.0E+00	9.0E+03	3.3E-04		2.2E+00	9.0E+03	2.5E-04	
NH3	1.4E+02	9.4E+02	1.5E-01		8.9E+00	9.4E+02	9.6E-03		6.6E+00	9.4E+02	7.1E-03	
SUMMARY HAZARD INDEX				1.0E+04	3.2E+01				5.1E+02			

TABLE R-38

## ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SETTLING POND 2, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Red fox			Red-tailed hawk		
	TBD	RTV	HI	TBD	RTV	HI
SO4	5.5E-03	1.2E+02	4.6E-05	7.6E-03	1.2E+02	6.3E-05
PB	4.4E-01	3.0E+00	1.5E-01	5.6E-01	2.5E+00	2.3E-01
24DNT	1.0E-02	1.0E+00	1.0E-02	1.6E-02	4.0E+01	4.1E-04
NIT	3.7E-03	1.3E+02	2.8E-05	5.1E-03	1.3E+02	3.8E-05
SN	7.0E-02	1.0E-01	7.0E-01	1.1E-01	3.5E+00	3.3E-02
DEP	1.6E-01	1.7E+02	9.5E-04	2.7E-01	1.7E+02	1.6E-03
DNBP	8.3E-04	6.0E+02	1.4E-06	1.4E-03	6.0E+02	2.3E-06
DPA	2.0E-03	2.5E+01	7.9E-05	3.2E-03	3.1E+01	1.0E-04
NC	2.4E-02	9.0E+03	2.7E-06	3.3E-02	9.0E+03	3.7E-06
NH3	7.2E-02	3.2E+02	2.3E-04	9.9E-02	9.4E+02	1.1E-04
SUMMARY HAZARD INDEX			8.5E-01	2.6E-01		

NOTES: TBD = Total Body Dose (mg/kgBW-day)

BW = Body Weight (kg)

RTV = Reference Toxicity Value (mg/kgBW-day)

HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-39

## ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SETTLING POND 3, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Short-tailed shrew</i>			<i>Eastern meadowlark</i>			<i>Garter snake</i>		
	TBD	RTV	HI	TBD	RTV	HI	TBD	RTV	HI
SO4	6.2E+00	1.2E+03	5.2E-03	3.8E-01	1.2E+03	3.2E-04	2.8E-01	1.2E+03	2.4E-04
PB	1.3E+02	2.0E+00	6.4E+01	7.1E+00	4.9E+00	1.4E+00	6.4E+00	2.0E+00	3.2E+00
24DNT	4.6E+00	5.4E+01	8.5E-02	2.8E-01	5.4E+01	5.3E-03	2.2E-01	5.4E+01	4.1E-03
26DNT	2.6E+00	5.4E+01	4.9E-02	1.6E-01	5.4E+01	3.0E-03	1.3E-01	5.4E+01	2.4E-03
NIT	8.4E-01	1.3E+03	6.3E-04	5.2E-02	1.3E+03	3.9E-05	3.9E-02	1.3E+03	2.9E-05
SN	1.3E+02	3.8E+01	3.4E+00	7.9E+00	3.5E+01	2.2E-01	6.1E+00	3.5E+01	1.8E-01
DEP	7.4E+01	1.7E+03	4.3E-02	4.4E+00	1.7E+03	2.5E-03	3.7E+00	1.7E+03	2.2E-03
DNBP	2.8E+01	6.0E+03	4.6E-03	1.5E+00	6.0E+03	2.6E-04	1.5E+00	6.0E+03	2.4E-04
DPA	4.9E+00	3.1E+02	1.6E-02	3.1E-01	3.1E+02	9.9E-04	2.4E-01	3.1E+02	7.7E-04
NC	3.3E+01	9.0E+04	3.6E-04	2.0E+00	9.0E+04	2.2E-05	1.5E+00	9.0E+04	1.7E-05
NH3	8.9E+01	3.2E+03	2.8E-02	5.5E+00	3.2E+03	1.7E-03	4.1E+00	3.2E+03	1.3E-03
SUMMARY HAZARD INDEX				6.8E+01		1.7E+00			3.4E+00



TABLE R-39  
ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SETTLING POND 3, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Red fox</i>			<i>Red-tailed hawk</i>		
	TBD	RTV	HI	TBD	RTV	HI
SO4	1.1E-01	1.2E+03	9.2E-05	3.0E-01	1.2E+03	2.5E-04
PB	2.1E+00	3.0E+01	7.1E-02	5.5E+00	2.5E+01	2.2E-01
24DNT	1.2E-01	5.0E+00	2.4E-02	4.0E-01	5.4E+01	7.4E-03
26DNT	7.0E-02	5.0E+00	1.4E-02	2.3E-01	5.4E+01	4.3E-03
NIT	1.5E-02	1.3E+03	1.1E-05	4.1E-02	1.3E+03	3.1E-05
SN	3.4E+00	3.8E+01	9.0E-02	1.1E+01	3.5E+01	3.2E-01
DEP	1.9E+00	1.7E+03	1.1E-03	6.3E+00	1.7E+03	3.7E-03
DNBP	6.9E-01	6.0E+03	1.2E-04	2.3E+00	6.0E+03	3.8E-04
DPA	1.3E-01	2.5E+02	5.3E-04	4.3E-01	3.1E+02	1.4E-03
NC	5.8E-01	9.0E+04	6.4E-06	1.6E+00	9.0E+04	1.8E-05
NH3	1.6E+00	3.2E+03	5.0E-04	4.4E+00	3.2E+03	1.4E-03
SUMMARY HAZARD INDEX			2.0E-01	5.5E-01		

NOTES: TBD = Total Body Dose (mg/kgBW-day)      BW = Body Weight (kg)  
RTV = Reference Toxicity Value (mg/kgBW-day)      HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-40  
ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SETTLING POND 3, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Short-tailed shrew			Eastern meadowlark			Garter snake		
	TBD	RTV	HI	TBD	RTV	HI	TBD	RTV	HI
SO4	6.2E+00	1.2E+02	5.2E-02	3.8E-01	1.2E+02	3.2E-03	2.8E-01	1.2E+02	2.4E-03
PB	1.3E+02	1.0E-01	1.3E+03	7.1E+00	1.8E+00	4.1E+00	6.4E+00	1.0E-01	6.4E+01
24DNT	4.6E+00	4.0E+01	1.1E-01	2.8E-01	4.0E+01	7.1E-03	2.2E-01	4.0E+01	5.5E-03
26DNT	2.6E+00	4.0E+01	6.6E-02	1.6E-01	4.0E+01	4.1E-03	1.3E-01	4.0E+01	3.2E-03
NIT	8.4E-01	1.3E+02	6.3E-03	5.2E-02	1.3E+02	3.9E-04	3.9E-02	1.3E+02	2.9E-04
SN	1.3E+02	1.0E-01	1.3E+03	7.9E+00	3.5E+00	2.2E+00	6.1E+00	1.0E-01	6.1E+01
DEP	7.4E+01	1.7E+02	4.3E-01	4.4E+00	1.7E+02	2.5E-02	3.7E+00	1.7E+02	2.2E-02
DNBP	2.8E+01	6.0E+02	4.6E-02	1.5E+00	6.0E+02	2.6E-03	1.5E+00	6.0E+02	2.4E-03
DPA	4.9E+00	3.1E+01	1.6E-01	3.1E-01	3.1E+01	9.9E-03	2.4E-01	3.1E+01	7.7E-03
NC	3.3E+01	9.0E+03	3.6E-03	2.0E+00	9.0E+03	2.2E-04	1.5E+00	9.0E+03	1.7E-04
NH3	8.9E+01	9.4E+02	9.5E-02	5.5E+00	9.4E+02	5.9E-03	4.1E+00	9.4E+02	4.4E-03
SUMMARY HAZARD INDEX			2.5E+03	6.4E+00			1.3E+02		

TABLE R-40  
ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION  
RF-MEDIAL INVESTIGATION  
SETTLING POND 3, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Red fox			Red-tailed hawk		
	TBD	RTV	HI	TBD	RTV	HI
SO4	1.1E-02	1.2E+02	9.2E-05	1.5E-02	1.2E+02	1.3E-04
PB	2.1E-01	3.0E+00	7.1E-02	2.7E-01	2.5E+00	1.1E-01
24DNT	1.2E-02	1.0E+00	1.2E-02	2.0E-02	4.0E+01	5.0E-04
26DNT	7.0E-03	1.0E+00	7.0E-03	1.2E-02	4.0E+01	2.9E-04
NIT	1.5E-03	1.3E+02	1.1E-05	2.1E-03	1.3E+02	1.6E-05
SN	3.4E-01	1.0E-01	3.4E+00	5.5E-01	3.5E+00	1.6E-01
DEP	1.9E-01	1.7E+02	1.1E-03	3.1E-01	1.7E+02	1.8E-03
DNBP	6.9E-02	6.0E+02	1.2E-04	1.2E-01	6.0E+02	1.9E-04
DPA	1.3E-02	2.5E+01	5.3E-04	2.1E-02	3.1E+01	6.9E-04
NC	5.8E-02	9.0E+03	6.4E-06	8.0E-02	9.0E+03	8.9E-06
NH3	1.6E-01	3.2E+02	5.0E-04	2.2E-01	9.4E+02	2.3E-04
SUMMARY HAZARD INDEX			3.5E+00	2.7E-01		

NOTES: TBD = Total Body Dose (mg/kgBW-day)      BW = Body Weight (kg)  
RTV = Reference Toxicity Value (mg/kgBW-day)      HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-41

## ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SETTLING POND 4, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Short-tailed shrew				Eastern meadowlark				Garter snake			
	TBD	RTV	HI		TBD	RTV	HI		TBD	RTV	HI	
SO4	6.9E+01	1.2E+03	5.7E-02		4.3E+00	1.2E+03	3.6E-03		3.2E+00	1.2E+03	2.6E-03	
PB	1.1E+03	2.0E+00	5.6E+02		6.3E+01	4.9E+00	1.3E+01		5.6E+01	2.0E+00	2.8E+01	
AL	1.1E+05	1.0E+03	1.1E+02		6.6E+03	1.0E+03	6.6E+00		5.1E+03	1.0E+03	5.1E+00	
NIT	1.7E+00	1.3E+03	1.3E-03		1.1E-01	1.3E+03	8.0E-05		7.9E-02	1.3E+03	5.9E-05	
SN	1.4E+02	3.8E+01	3.6E+00		8.4E+00	3.5E+01	2.4E-01		6.6E+00	3.5E+01	1.9E-01	
DPA	6.3E-01	3.1E+02	2.0E-03		3.9E-02	3.1E+02	1.3E-04		3.1E-02	3.1E+02	9.9E-05	
NC	1.8E+02	9.0E+04	2.0E-03		1.1E+01	9.0E+04	1.2E-04		8.2E+00	9.0E+04	9.1E-05	
NH3	1.6E+02	3.2E+03	5.2E-02		1.0E+01	3.2E+03	3.2E-03		7.6E+00	3.2E+03	2.4E-03	
SUMMARY HAZARD INDEX				6.7E+02			2.0E+01				3.3E+01	

TABLE R-41  
ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SETTLING POND 4, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Red fox</i>			<i>Red-tailed hawk</i>		
	TBD	RTV	HI	TBD	RTV	HI
SO4	1.2E+00	1.2E+03	1.0E-03	3.4E+00	1.2E+03	2.8E-03
PB	1.9E+01	3.0E+01	6.2E-01	4.8E+01	2.5E+01	1.9E+00
AL	2.8E+03	1.0E+03	2.8E+00	9.2E+03	1.0E+03	9.2E+00
NIT	3.1E-02	1.3E+03	2.3E-05	8.4E-02	1.3E+03	6.3E-05
SN	3.6E+00	3.8E+01	9.6E-02	1.2E+01	3.5E+01	3.4E-01
DPA	1.7E-02	2.5E+02	6.8E-05	5.5E-02	3.1E+02	1.8E-04
NC	3.2E+00	9.0E+04	3.5E-05	8.8E+00	9.0E+04	9.7E-05
NH3	2.9E+00	3.2E+03	9.2E-04	8.1E+00	3.2E+03	2.5E-03
SUMMARY HAZARD INDEX			3.5E+00	1.1E+01		

NOTES: TBD = Total Body Dose (mg/kgBW-day)      BW = Body Weight (kg)  
RTV = Reference Toxicity Value (mg/kgBW-day)      HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-42

## ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SETTLING POND 4, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Short-tailed shrew</i>			<i>Eastern meadowlark</i>			<i>Garter snake</i>		
	TBD	RTV	HI	TBD	RTV	HI	TBD	RTV	HI
SO4	6.9E+01	1.2E+02	5.7E-01	4.3E+00	1.2E+02	3.5E-02	3.2E+00	1.2E+02	2.6E-02
PB	1.1E+03	1.0E-01	1.1E+04	6.3E+01	1.8E+00	3.6E+01	5.6E+01	1.0E-01	5.6E+02
AL	1.1E+05	1.0E+02	1.1E+03	6.6E+03	1.0E+02	6.6E+01	5.1E+03	1.0E+02	5.1E+01
NIT	1.7E+00	1.3E+02	1.3E-02	1.1E-01	1.3E+02	8.0E-04	7.9E-02	1.3E+02	5.9E-04
SN	1.4E+02	1.0E-01	1.4E+03	8.4E+00	3.5E+00	2.4E+00	6.6E+00	1.0E-01	6.6E+01
DPA	6.3E-01	3.1E+01	2.0E-02	3.9E-02	3.1E+01	1.3E-03	3.1E-02	3.1E+01	9.9E-04
NC	1.8E+02	9.0E+03	2.0E-02	1.1E+01	9.0E+03	1.2E-03	8.2E+00	9.0E+03	9.1E-04
NH3	1.6E+02	9.4E+02	1.8E-01	1.0E+01	9.4E+02	1.1E-02	7.6E+00	9.4E+02	8.1E-03
SUMMARY HAZARD INDEX			1.4E+04			1.0E+02			6.8E+02

TABLE R-42  
ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SETTLING POND 4, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Red fox			Red-tailed hawk		
	TBD	RTV	HI	TBD	RTV	HI
SO4	9.8E-02	1.2E+02	8.1E-04	1.3E-01	1.2E+02	1.1E-03
P8	1.5E+00	3.0E+00	5.0E-01	1.9E+00	2.5E+00	7.7E-01
AL	2.3E+02	1.0E+02	2.3E+00	3.7E+02	1.0E+02	3.7E+00
NIT	2.4E-03	1.3E+02	1.8E-05	3.4E-03	1.3E+02	2.5E-05
SN	2.9E-01	1.0E-01	2.9E+00	4.7E-01	3.5E+00	1.3E-01
DPA	1.4E-03	2.5E+01	5.4E-05	2.2E-03	3.1E+01	7.1E-05
NC	2.5E-01	9.0E+03	2.8E-05	3.5E-01	9.0E+03	3.9E-05
NH3	2.3E-01	3.2E+02	7.4E-04	3.2E-01	9.4E+02	3.5E-04
SUMMARY HAZARD INDEX			5.6E+00	4.6E+00		

NOTES: TBD = Total Body Dose (mg/kgBW-day)      BW = Body Weight (kg)  
RTV = Reference Toxicity Value (mg/kgBW-day)      HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-43

## ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SPOILS DISPOSAL AREA 1, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Short-tailed shrew				Eastern meadowlark				Garter snake			
	TBD	RTV	HI		TBD	RTV	HI		TBD	RTV	HI	
SO4	2.5E+01	1.2E+03	2.1E-02		1.6E+00	1.2E+03	1.3E-03		1.2E+00	1.2E+03	9.6E-04	
PB	1.3E+03	2.0E+00	6.6E+02		7.3E+01	4.9E+00	1.5E+01		6.5E+01	2.0E+00	3.3E+01	
24DNT	2.1E+01	5.4E+01	3.9E-01		1.3E+00	5.4E+01	2.4E-02		1.0E+00	5.4E+01	1.9E-02	
26DNT	1.8E+00	5.4E+01	3.3E-02		1.1E-01	5.4E+01	2.0E-03		8.5E-02	5.4E+01	1.6E-03	
NIT	2.7E+00	1.3E+03	2.1E-03		1.7E-01	1.3E+03	1.3E-04		1.3E-01	1.3E+03	9.5E-05	
SN	6.5E+00	3.8E+01	1.7E-01		4.0E-01	3.5E+01	1.1E-02		3.1E-01	3.5E+01	9.0E-03	
ZN	2.7E+03	5.0E+02	5.4E+00		1.7E+02	5.0E+02	3.5E-01		2.1E+02	5.0E+02	4.3E-01	
DNBP	8.1E+01	6.0E+03	1.4E-02		4.5E+00	6.0E+03	7.5E-04		4.3E+00	6.0E+03	7.1E-04	
DPA	4.2E+01	3.1E+02	1.4E-01		2.6E+00	3.1E+02	8.5E-03		2.0E+00	3.1E+02	6.6E-03	
NC	1.9E+03	9.0E+04	2.1E-02		1.2E+02	9.0E+04	1.3E-03		8.7E+01	9.0E+04	9.6E-04	
CH2Cl2	1.8E-02	5.3E+02	3.3E-05		1.1E-03	5.3E+02	2.1E-06		8.5E-04	5.3E+02	1.6E-06	
B2EHP	5.6E-01	1.7E+03	3.2E-04		3.1E-02	1.7E+03	1.8E-05		2.9E-02	1.7E+03	1.7E-05	
NG	3.3E+01	3.2E+02	1.1E-01		2.1E+00	3.2E+02	6.6E-03		1.6E+00	3.2E+02	5.1E-03	
CL	3.3E+00	6.0E+02	5.4E-03		2.0E-01	6.0E+02	3.4E-04		1.5E-01	6.0E+02	2.5E-04	
BR	2.1E+00	7.0E+02	2.9E-03		1.3E-01	7.0E+02	1.8E-04		9.5E-02	7.0E+02	1.4E-04	
DNOP	1.4E+01	1.8E+03	7.8E-03		7.6E-01	1.8E+03	4.3E-04		7.2E-01	1.8E+03	4.1E-04	
SUMMARY HAZARD INDEX				6.6E+02								



TABLE R-43  
ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 1, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Red fox			Red-tailed hawk		
	TBD	RTV	HI	TBD	RTV	HI
SO4	4.5E-01	1.2E+03	3.7E-04	1.2E+00	1.2E+03	1.0E-03
PB	2.2E+01	3.0E+01	7.2E-01	5.6E+01	2.5E+01	2.3E+00
24DNT	5.6E-01	5.0E+00	1.1E-01	1.8E+00	5.4E+01	3.4E-02
26DNT	4.7E-02	5.0E+00	9.4E-03	1.5E-01	5.4E+01	2.8E-03
NIT	4.9E-02	1.3E+03	3.7E-05	1.3E-01	1.3E+03	1.0E-04
SN	1.7E-01	3.8E+01	4.6E-03	5.6E-01	3.5E+01	1.6E-02
ZN	4.2E+02	5.0E+02	8.5E-01	1.6E+03	5.0E+02	3.1E+00
DNBP	2.0E+00	6.0E+03	3.4E-04	6.7E+00	6.0E+03	1.1E-03
DPA	1.1E+00	2.5E+02	4.5E-03	3.7E+00	3.1E+02	1.2E-02
NC	3.4E+01	9.0E+04	3.7E-04	9.3E+01	9.0E+04	1.0E-03
CH2Cl2	4.7E-04	5.3E+02	8.9E-07	1.5E-03	5.3E+02	2.9E-06
B2EHP	1.4E-02	1.7E+03	8.1E-06	4.6E-02	1.7E+03	2.7E-05
NG	8.9E-01	2.5E+01	3.6E-02	2.9E+00	3.2E+02	9.2E-03
CL	5.8E-02	6.0E+02	9.7E-05	1.6E-01	6.0E+02	2.7E-04
BR	3.7E-02	7.0E+02	5.2E-05	1.0E-01	7.0E+02	1.4E-04
DNOP	3.4E-01	1.8E+03	1.9E-04	1.1E+00	1.8E+03	6.5E-04
SUMMARY HAZARD INDEX			1.7E+00	5.5E+00		

NOTES: TBD = Total Body Dose (mg/kgBW-day)      BW = Body Weight (kg)  
RTV = Reference Toxicity Value (mg/kgBW-day)      HI = Hazard Index (calculated by dividing TRD by RTV)

TABLE R-44  
ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 1, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Short-tailed shrew			Eastern meadowlark			Garter snake		
	TBD	RTV	HI	TBD	RTV	HI	TBD	RTV	HI
SO4	2.5E+01	1.2E+02	2.1E-01	1.6E+00	1.2E+02	1.3E-02	1.2E+00	1.2E+02	9.6E-03
PB	1.3E+03	1.0E-01	1.3E+04	7.3E+01	1.8E+00	4.2E+01	6.5E+01	1.0E-01	6.5E+02
24DNT	2.1E+01	4.0E+01	5.3E-01	1.3E+00	4.0E+01	3.3E-02	1.0E+00	4.0E+01	2.6E-02
26DNT	1.8E+00	4.0E+01	4.4E-02	1.1E-01	4.0E+01	2.7E-03	8.5E-02	4.0E+01	2.1E-03
NIT	2.7E+00	1.3E+02	2.1E-02	1.7E-01	1.3E+02	1.3E-03	1.3E-01	1.3E+02	9.5E-04
SN	6.5E+00	1.0E-01	6.5E+01	4.0E-01	3.5E+00	1.1E-01	3.1E-01	1.0E-01	3.1E+00
ZN	2.7E+03	1.6E+02	1.7E+01	1.7E+02	1.6E+02	1.1E+00	2.1E+02	1.6E+02	1.3E+00
DNBP	8.1E+01	6.0E+02	1.4E-01	4.5E+00	6.0E+02	7.5E-03	4.3E+00	6.0E+02	7.1E-03
DPA	4.2E+01	3.1E+01	1.4E+00	2.6E+00	3.1E+01	8.5E-02	2.0E+00	3.1E+01	6.6E-02
NC	1.9E+03	9.0E+03	2.1E-01	1.2E+02	9.0E+03	1.3E-02	8.7E+01	9.0E+03	9.6E-03
CH2Cl2	1.8E-02	5.3E+01	3.3E-04	1.1E-03	5.3E+01	2.1E-05	8.5E-04	5.3E+01	1.6E-05
B2EHP	5.6E-01	1.9E+01	2.9E-02	3.1E-02	1.9E+01	1.6E-03	2.9E-02	1.9E+01	1.5E-03
NG	3.3E+01	3.2E+01	1.1E+00	2.1E+00	3.2E+01	6.6E-02	1.6E+00	3.2E+01	5.1E-02
CL	3.3E+00	6.0E+01	5.4E-02	2.0E-01	6.0E+01	3.4E-03	1.5E-01	6.0E+01	2.5E-03
BR	2.1E+00	7.0E+01	2.9E-02	1.3E-01	7.0E+01	1.8E-03	9.5E-02	7.0E+01	1.4E-03
DNOP	1.4E+01	1.8E+02	7.8E-02	7.6E-01	1.8E+02	4.3E-03	7.2E-01	1.8E+02	4.1E-03
SUMMARY HAZARD INDEX			1.3E+04			4.3E+01			6.6E+02

TABLE R-44

## ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SPOILS DISPOSAL AREA 1, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Red fox			Red-tailed hawk		
	TBD	RTV	HI	TBD	RTV	HI
SO4	8.9E-03	1.2E+02	7.4E-05	1.2E-02	1.2E+02	1.0E-04
PB	4.3E-01	3.0E+00	1.4E-01	5.6E-01	2.5E+00	2.3E-01
24DNT	1.1E-02	1.0E+00	1.1E-02	1.8E-02	4.0E+01	4.6E-04
26DNT	9.4E-04	1.0E+00	9.4E-04	1.5E-03	4.0E+01	3.8E-05
NIT	9.8E-04	1.3E+02	7.3E-06	1.3E-03	1.3E+02	1.0E-05
SN	3.5E-03	1.0E-01	3.5E-02	5.6E-03	3.5E+00	1.6E-03
ZN	8.5E+00	1.6E+02	5.3E-02	1.6E+01	1.6E+02	9.8E-02
DNBP	4.1E-02	6.0E+02	6.8E-05	6.7E-02	6.0E+02	1.1E-04
DPA	2.3E-02	2.5E+01	9.0E-04	3.7E-02	3.1E+01	1.2E-03
NC	6.7E-01	9.0E+03	7.5E-05	9.3E-01	9.0E+03	1.0E-04
CH2Cl2	9.4E-06	5.3E+01	1.8E-07	1.5E-05	5.3E+01	2.9E-07
B2EHP	2.8E-04	1.9E+01	1.5E-05	4.6E-04	1.9E+01	2.4E-05
NG	1.8E-02	3.0E+00	5.9E-03	2.9E-02	3.2E+01	9.2E-04
CL	1.2E-03	6.0E+01	1.9E-05	1.6E-03	6.0E+01	2.7E-05
BR	7.3E-04	7.0E+01	1.0E-05	1.0E-03	7.0E+01	1.4E-05
DNOP	6.8E-03	1.8E+02	3.9E-05	1.1E-02	1.8E+02	6.5E-05
SUMMARY HAZARD INDEX			2.5E-01			3.3E-01

NOTES: TBD = Total Body Dose (mg/kgBW-day)

BW = Body Weight (kg)

RTV = Reference Toxicity Value (mg/kgBW-day)

HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-45

ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

SPOILS DISPOSAL AREA 2, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Short-tailed shrew			Eastern meadowlark			Garter snake		
	TBD	RTV	HI	TBD	RTV	HI	TBD	RTV	HI
SO4	2.2E+01	1.2E+03	1.9E-02	1.4E+00	1.2E+03	1.2E-03	1.0E+00	1.2E+03	8.6E-04
PB	1.4E+03	2.0E+00	7.0E+02	7.8E+01	4.9E+00	1.6E+01	6.9E+01	2.0E+00	3.5E+01
NIT	1.7E+00	1.3E+03	1.3E-03	1.1E-01	1.3E+03	8.0E-05	7.9E-02	1.3E+03	5.9E-05
SN	7.1E+00	3.8E+01	1.9E-01	4.4E-01	3.5E+01	1.3E-02	3.4E-01	3.5E+01	9.7E-03
ZN	9.6E+03	5.0E+02	1.9E+01	6.2E+02	5.0E+02	1.2E+00	6.9E+02	5.0E+02	1.4E+00
DNBP	9.3E+00	6.0E+03	1.5E-03	5.1E-01	6.0E+03	8.6E-05	4.8E-01	6.0E+03	8.0E-05
DPA	5.6E+00	3.1E+02	1.8E-02	3.5E-01	3.1E+02	1.1E-03	2.7E-01	3.1E+02	8.7E-04
NC	1.4E+03	9.0E+04	1.5E-02	8.5E+01	9.0E+04	9.5E-04	6.3E+01	9.0E+04	7.0E-04
CH2Cl2	2.1E-02	5.3E+02	4.0E-05	1.3E-03	5.3E+02	2.5E-06	1.0E-03	5.3E+02	1.9E-06
BR	6.9E-01	7.0E+02	9.8E-04	4.3E-02	7.0E+02	6.1E-05	3.2E-02	7.0E+02	4.5E-05
CL	4.0E+00	6.0E+02	6.6E-03	2.4E-01	6.0E+02	4.1E-04	1.8E-01	6.0E+02	3.0E-04
24DNT	2.3E+00	5.4E+01	4.2E-02	1.4E-01	5.4E+01	1.6E-03	1.1E-01	5.4E+01	2.0E-03
SUMMARY HAZARD INDEX				7.2E+02		1.7E+01			3.6E+01

AD-A280 442

REMEDIAL INVESTIGATION BADGER ARMY AMMUNITION PLANT  
BARABOO WISCONSIN VOLUME 7 APPENDICES M THROUGH R(U)  
ABB ENVIRONMENTAL PORTLAND ME 1991 XA-USATHAMA  
DAAA15-91-D-0008

10710

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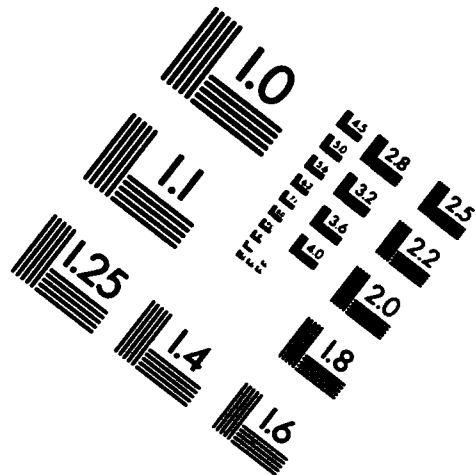
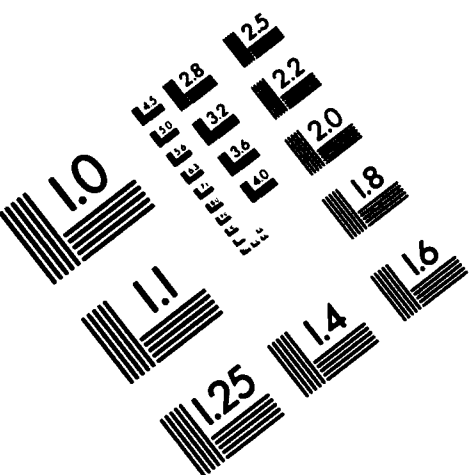


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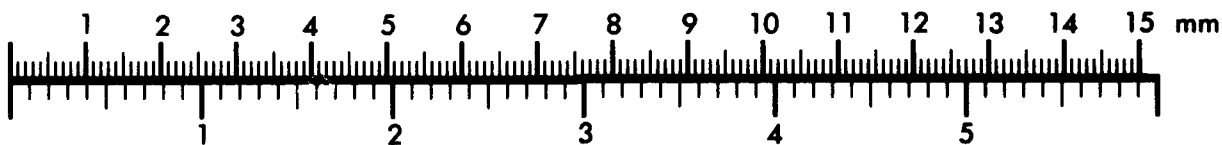
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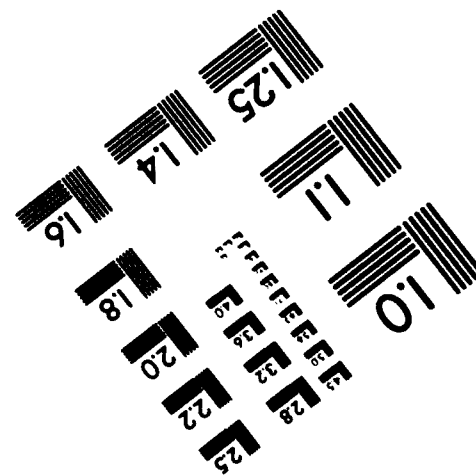
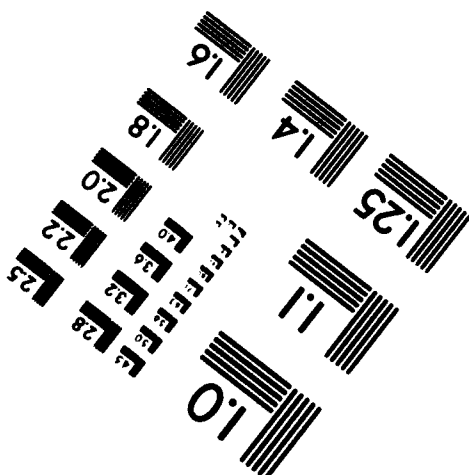
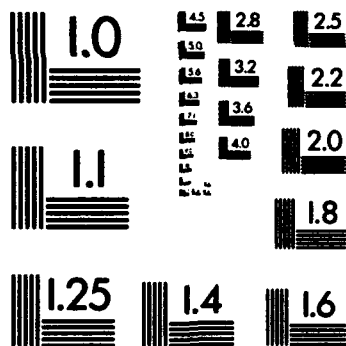
301/587-8202



**Centimeter**



**Inches**



MANUFACTURED TO AIIM STANDARDS  
BY APPLIED IMAGE, INC.

TABLE R-45  
ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 2, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Red fox</i>			<i>Red-tailed hawk</i>		
	TBD	RTV	HI	TBD	RTV	HI
SO4	4.0E-01	1.2E+03	3.3E-04	1.1E+00	1.2E+03	9.2E-04
PB	2.1E+01	3.0E+01	7.0E-01	5.4E+01	2.5E+01	2.2E+00
NIT	3.1E-02	1.3E+03	2.3E-05	8.4E-02	1.3E+03	6.3E-05
SN	1.8E-01	3.8E+01	4.7E-03	5.6E-01	3.5E+01	1.6E-02
ZN	1.2E+03	5.0E+02	2.4E+00	4.5E+03	5.0E+02	9.0E+00
DNBP	2.1E-01	6.0E+03	3.5E-05	7.0E-01	6.0E+03	1.2E-04
DPA	1.4E-01	2.5E+02	5.5E-04	4.5E-01	3.1E+02	1.4E-03
NC	2.4E+01	9.0E+04	2.7E-04	6.7E+01	9.0E+04	7.5E-04
CH2Cl2	5.2E-04	5.3E+02	9.9E-07	1.7E-03	5.3E+02	3.2E-06
BR	1.2E-02	7.0E+02	1.7E-05	3.4E-02	7.0E+02	4.8E-05
CL	7.0E-02	6.0E+02	1.2E-04	1.9E-01	6.0E+02	3.2E-04
24DNT	5.6E-02	5.0E+00	1.1E-02	1.8E-01	5.4E+01	3.4E-03
SUMMARY HAZARD INDEX			3.1E+00	1.1E+01		

NOTES: TBD = Total Body Dose (mg/kgBW-day)      BW = Body Weight (kg)  
RTV = Reference Toxicity Value (mg/kgBW-day)      HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-46

## ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SPOILS DISPOSAL AREA 2, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Short-tailed shrew			Eastern meadowlark			Garter snake		
	TBD	RTV	HI	TBD	RTV	HI	TBD	RTV	HI
SO4	2.2E+01	1.2E+02	1.9E-01	9.7E-01	1.2E+02	8.1E-03	7.2E-01	1.2E+02	6.0E-03
PB	1.4E+03	1.0E-01	1.4E+04	5.5E+01	1.8E+00	3.1E+01	4.9E+01	1.0E-01	4.9E+02
NIT	1.7E+00	1.3E+02	1.3E-02	7.5E-02	1.3E+02	5.6E-04	5.5E-02	1.3E+02	4.1E-04
SN	7.1E+00	1.0E-01	7.1E+01	3.1E-01	3.5E+00	8.8E-02	2.4E-01	1.0E-01	2.4E+00
ZN	9.6E+03	1.6E+02	6.0E+01	4.3E+02	1.6E+02	2.7E+00	4.8E+02	1.6E+02	3.0E+00
DNBP	9.3E+00	6.0E+02	1.5E-02	3.6E-01	6.0E+02	6.0E-04	3.4E-01	6.0E+02	5.6E-04
DPA	5.6E+00	3.1E+01	1.8E-01	2.4E-01	3.1E+01	7.9E-03	1.9E-01	3.1E+01	6.1E-03
NC	1.4E+03	9.0E+03	1.5E-01	6.0E+01	9.0E+03	6.6E-03	4.4E+01	9.0E+03	4.9E-03
CH2Cl2	2.1E-02	5.3E+01	4.0E-04	9.2E-04	5.3E+01	1.7E-05	7.0E-04	5.3E+01	1.3E-05
BR	6.9E-01	7.0E+01	9.8E-03	3.0E-02	7.0E+01	4.3E-04	2.2E-02	7.0E+01	3.2E-04
CL	4.0E+00	6.0E+01	6.6E-02	1.7E-01	6.0E+01	2.9E-03	1.3E-01	6.0E+01	2.1E-03
24DNT	2.3E+00	4.0E+01	5.7E-02	9.9E-02	4.0E+01	2.5E-03	7.6E-02	4.0E+01	1.9E-03
SUMMARY HAZARD INDEX			1.4E+04			3.4E+01			4.9E+02



TABLE R-46  
ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 2, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Red fox			Red-tailed hawk		
	TBD	RTV	HI	TBD	RTV	HI
SO4	5.6E-03	1.2E+02	4.6E-05	7.7E-03	1.2E+02	6.4E-05
PB	3.0E-01	3.0E+00	9.8E-02	3.8E-01	2.5E+00	1.5E-01
PIT	4.3E-04	1.3E+02	3.2E-06	5.9E-04	1.3E+02	4.4E-06
SN	2.5E-03	1.0E-01	2.5E-02	3.9E-03	3.5E+00	1.1E-03
ZN	1.7E+01	1.6E+02	1.0E-01	3.2E+01	1.6E+02	2.0E-01
DNBP	3.0E-03	6.0E+02	4.9E-06	4.9E-03	6.0E+02	8.1E-06
DPA	1.9E-03	2.5E+01	7.8E-05	3.1E-03	3.1E+01	1.0E-04
NC	3.4E-01	9.0E+03	3.8E-05	4.7E-01	9.0E+03	5.2E-05
CH2Cl2	7.3E-06	5.3E+01	1.4E-07	1.2E-05	5.3E+01	2.2E-07
BR	1.7E-04	7.0E+01	2.4E-06	2.4E-04	7.0E+01	3.4E-06
CL	9.8E-04	6.0E+01	1.6E-05	1.4E-03	6.0E+01	2.3E-05
24DNT	7.9E-04	1.0E+00	7.9E-04	1.3E-03	4.0E+01	3.2E-05
SUMMARY HAZARD INDEX			2.3E-01	3.5E-01		

NOTES: TBD = Total Body Dose (mg/kgBW-day)      BW = Body Weight (kg)  
RTV = Reference Toxicity Value (mg/kgBW-day)      HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-47

## ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SPOILS DISPOSAL AREA 3, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Short-tailed shrew			Eastern meadowlark			Garter snake		
	TBD	RTV	HI	TBD	RTV	HI	TBD	RTV	HI
SO4	1.3E+01	1.2E+03	1.1E-02	8.0E-01	1.2E+03	6.7E-04	5.9E-01	1.2E+03	4.9E-04
PB	2.5E+02	2.0E+00	1.3E+02	1.4E+01	4.9E+00	2.9E+00	1.2E+01	2.0E+00	6.2E+00
NIT	3.8E+00	1.3E+03	2.8E-03	2.3E-01	1.3E+03	1.8E-04	1.7E-01	1.3E+03	1.3E-04
SN	1.0E+01	3.8E+01	2.7E-01	6.3E-01	3.5E+01	1.8E-02	4.8E-01	3.5E+01	1.4E-02
ZN	3.2E+03	5.0E+02	6.4E+00	2.1E+02	5.0E+02	4.1E-01	2.2E+02	5.0E+02	4.4E-01
DNBP	6.4E+00	6.0E+03	1.1E-03	3.6E-01	6.0E+03	5.9E-05	3.3E-01	6.0E+03	5.5E-05
DPA	3.9E+00	3.1E+02	1.3E-02	2.4E-01	3.1E+02	7.7E-04	1.8E-01	3.1E+02	5.9E-04
NC	6.5E+02	9.0E+04	7.3E-03	4.0E+01	9.0E+04	4.5E-04	3.0E+01	9.0E+04	3.3E-04
CH2Cl2	4.4E-02	5.3E+02	8.4E-05	2.7E-03	5.3E+02	5.2E-06	2.1E-03	5.3E+02	4.0E-06
CL	2.9E+00	6.0E+02	4.9E-03	1.8E-01	6.0E+02	3.0E-04	1.3E-01	6.0E+02	2.2E-04
24DNT	1.9E+00	5.4E+01	3.6E-02	1.2E-01	5.4E+01	2.2E-03	9.2E-02	5.4E+01	1.7E-03
SUMMARY HAZARD INDEX			1.3E+02	3.3E+00			6.7E+00		

TABLE R-47  
ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 3, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Red fox</i>			<i>Red-tailed hawk</i>		
	TBD	RTV	HI	TBD	RTV	HI
SO4	2.3E-01	1.2E+03	1.9E-04	6.3E-01	1.2E+03	5.3E-04
PB	3.7E+00	3.0E+01	1.2E-01	9.3E+00	2.5E+01	3.7E-01
NIT	6.7E-02	1.3E+03	5.0E-05	1.9E-01	1.3E+03	1.4E-04
SN	2.4E-01	3.8E+01	6.5E-03	7.8E-01	3.5E+01	2.2E-02
ZN	3.7E+02	5.0E+02	7.3E-01	1.4E+03	5.0E+02	2.8E+00
DNBP	1.4E-01	6.0E+03	2.4E-05	4.6E-01	6.0E+03	7.7E-05
DPA	9.3E-02	2.5E+02	3.7E-04	3.0E-01	3.1E+02	9.6E-04
NC	1.2E+01	9.0E+04	1.3E-04	3.2E+01	9.0E+04	3.6E-04
CH2Cl2	1.1E-03	5.3E+02	2.0E-06	3.4E-03	5.3E+02	6.4E-06
CL	5.2E-02	6.0E+02	8.6E-05	1.4E-01	6.0E+02	2.4E-04
24DNT	4.6E-02	5.0E+00	9.3E-03	1.5E-01	5.4E+01	2.7E-03
SUMMARY HAZARD INDEX			8.7E-01	3.2E+00		

NOTES: TBD = Total Body Dose (mg/kgBW-day)      BW = Body Weight (kg)  
RTV = Reference Toxicity Value (mg/kgBW-day)      HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-48  
ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 3, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Short-tailed shrew			Eastern meadowlark			Garter snake		
	TBD	RTV	HI	TBD	RTV	HI	TBD	RTV	HI
SO4	1.3E+01	1.2E+02	1.1E-01	4.8E-01	1.2E+02	4.0E-03	3.5E-01	1.2E+02	3.0E-03
PB	2.5E+02	1.0E-01	2.5E+03	8.4E+00	1.8E+00	4.8E+00	7.5E+00	1.0E-01	7.5E+01
NIT	3.8E+00	1.3E+02	2.8E-02	1.4E-01	1.3E+02	1.1E-03	1.0E-01	1.3E+02	7.8E-04
SN	1.0E+01	1.0E-01	1.0E+02	3.8E-01	3.5E+00	1.1E-01	2.9E-01	1.0E-01	2.9E+00
ZN	3.2E+03	1.6E+02	2.0E+01	1.2E+02	1.6E+02	7.7E-01	1.3E+02	1.6E+02	8.3E-01
DNBP	6.4E+00	6.0E+02	1.1E-02	2.1E-01	6.0E+02	3.6E-04	2.0E-01	6.0E+02	3.3E-04
DPA	3.9E+00	3.1E+01	1.3E-01	1.4E-01	3.1E+01	4.6E-03	1.1E-01	3.1E+01	3.6E-03
NC	6.5E+02	9.0E+03	7.3E-02	2.4E+01	9.0E+03	2.7E-03	1.8E+01	9.0E+03	2.0E-03
CH2Cl2	4.4E-02	5.3E+01	8.4E-04	1.6E-03	5.3E+01	3.1E-05	1.3E-03	5.3E+01	2.4E-05
CL	2.9E+00	6.0E+01	4.9E-02	1.1E-01	6.0E+01	1.8E-03	8.0E-02	6.0E+01	1.3E-03
24DNT	1.9E+00	4.0E+01	4.8E-02	7.2E-02	4.0E+01	1.8E-03	5.5E-02	4.0E+01	1.4E-03
SUMMARY HAZARD INDEX			2.6E+03	5.7E+00			7.9E+01		

TABLE R-48

## ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SPOILS DISPOSAL AREA 3, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Red fox</i>			<i>Red-tailed hawk</i>		
	TBD	RTV	HI	TBD	RTV	HI
SO4	2.7E-03	1.2E+02	2.3E-05	3.8E-03	1.2E+02	3.2E-05
PB	4.4E-02	3.0E+00	1.5E-02	5.6E-02	2.5E+00	2.2E-02
NIT	8.1E-04	1.3E+02	6.1E-06	1.1E-03	1.3E+02	8.4E-06
SN	2.9E-03	1.0E-01	2.9E-02	4.7E-03	3.5E+00	1.3E-03
ZN	4.4E+00	1.6E+02	2.8E-02	8.4E+00	1.6E+02	5.3E-02
DNBP	1.7E-03	6.0E+02	2.8E-06	2.8E-03	6.0E+02	4.6E-06
DPA	1.1E-03	2.5E+01	4.5E-05	1.8E-03	3.1E+01	5.7E-05
NC	1.4E-01	9.0E+03	1.5E-05	1.9E-01	9.0E+03	2.1E-05
CH2Cl2	1.3E-05	5.3E+01	2.4E-07	2.0E-05	5.3E+01	3.8E-07
CL	6.2E-04	6.0E+01	1.0E-05	8.6E-04	6.0E+01	1.4E-05
24DNT	5.6E-04	1.0E+00	5.6E-04	8.9E-04	4.0E+01	2.2E-05
SUMMARY HAZARD INDEX			7.2E-02			
						7.7E-02

NOTES: TBD = Total Body Dose (mg/kgBW-day)

BW = Body Weight (kg)

RTV = Reference Toxicity Value (mg/kgBW-day)

HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-49

## ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SPOILS DISPOSAL AREA 4, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Short-tailed shrew</i>			<i>Eastern meadowlark</i>			<i>Garter snake</i>		
	TBD	RTV	HI	TBD	RTV	HI	TBD	RTV	HI
SO4	2.4E+01	1.2E+03	2.0E-02	1.5E+00	1.2E+03	1.2E-03	1.1E+00	1.2E+03	9.1E-04
PB	4.5E+02	2.0E+00	2.3E+02	2.5E+01	4.9E+00	5.1E+00	2.2E+01	2.0E+00	1.1E+01
24DNT	1.2E+00	5.4E+01	2.3E-02	7.6E-02	5.4E+01	1.4E-03	6.0E-02	5.4E+01	1.1E-03
NIT	2.1E+00	1.3E+03	1.5E-03	1.3E-01	1.3E+03	9.6E-05	9.5E-02	1.3E+03	7.1E-05
SN	2.9E+00	3.8E+01	7.7E-02	1.8E-01	3.5E+01	5.1E-03	1.4E-01	3.5E+01	4.0E-03
ZN	2.6E+03	5.0E+02	5.2E+00	1.7E+02	5.0E+02	3.4E-01	2.1E+02	5.0E+02	4.1E-01
DNBP	7.0E+00	6.0E+03	1.2E-03	3.9E-01	6.0E+03	6.5E-05	3.7E-01	6.0E+03	6.2E-05
DPA	1.9E+00	3.1E+02	6.3E-03	1.2E-01	3.1E+02	3.9E-04	9.4E-02	3.1E+02	3.0E-04
NC	5.2E+02	9.0E+04	5.7E-03	3.2E+01	9.0E+04	3.5E-04	2.4E+01	9.0E+04	2.6E-04
CH2Cl2	1.8E-02	5.3E+02	3.3E-05	1.1E-03	5.3E+02	2.1E-06	8.5E-04	5.3E+02	1.6E-06
B2EHP	5.1E-01	1.7E+03	3.0E-04	2.8E-02	1.7E+03	1.6E-05	2.7E-02	1.7E+03	1.6E-05
CL	2.2E+00	6.0E+02	3.7E-03	1.4E-01	6.0E+02	2.3E-04	1.0E-01	6.0E+02	1.7E-04
DNOP	1.0E+00	1.8E+03	5.7E-04	5.6E-02	1.8E+03	3.2E-05	5.3E-02	1.8E+03	3.0E-05
SUMMARY HAZARD INDEX	2.3E+02			5.5E+00			1.2E+01		

TABLE R-49  
ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 4, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Red fox			Red-tailed hawk		
	TBD	RTV	HI	TBD	RTV	HI
SO4	4.2E-01	1.2E+03	3.5E-04	1.2E+00	1.2E+03	9.8E-04
78	7.5E+00	3.0E+01	2.5E-01	1.9E+01	2.5E+01	7.7E-01
24DNT	3.3E-02	5.0E+00	6.6E-03	1.1E-01	5.4E+01	2.0E-03
NIT	3.7E-02	1.3E+03	2.8E-05	1.0E-01	1.3E+03	7.6E-05
SN	7.7E-02	3.8E+01	2.0E-03	2.5E-01	3.5E+01	7.2E-03
ZN	4.1E+02	5.0E+02	8.2E-01	1.5E+03	5.0E+02	3.0E+00
DNBP	1.8E-01	6.0E+03	2.9E-05	5.8E-01	6.0E+03	9.7E-05
DPA	5.2E-02	2.5E+02	2.1E-04	1.7E-01	3.1E+02	5.4E-04
NC	9.2E+00	9.0E+04	1.0E-04	2.5E+01	9.0E+04	2.8E-04
CH2Cl2	4.7E-04	5.3E+02	8.9E-07	1.5E-03	5.3E+02	2.9E-06
B2EHP	1.3E-02	1.7E+03	7.4E-06	4.2E-02	1.7E+03	2.5E-05
CL	4.0E-02	6.0E+02	6.6E-05	1.1E-01	6.0E+02	1.8E-04
DNOP	2.5E-02	1.8E+03	1.4E-05	8.3E-02	1.8E+03	4.7E-05
SUMMARY HAZARD INDEX			1.1E+00	3.8E+00		

NOTES: TBD = Total Body Dose (mg/kgBW-day)      BW = Body Weight (kg)  
RTV = Reference Toxicity Value (mg/kgBW-day)      HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-50

## ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SPOILS DISPOSAL AREA 4, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Short-tailed shrew				Eastern meadowlark				Garter snake			
	TBD	RTV	HI		TBD	RTV	HI		TBD	RTV	HI	
SO4	2.4E+01	1.2E+02	2.0E-01		1.5E+00	1.2E+02	1.2E-02		1.1E+00	1.2E+02	9.1E-03	
PB	4.5E+02	1.0E-01	4.5E+03		2.5E+01	1.8E+00	1.4E+01		2.2E+01	1.0E-01	2.2E+02	
24DNT	1.2E+00	4.0E+01	3.1E-02		7.6E-02	4.0E+01	1.9E-03		6.0E-02	4.0E+01	1.5E-03	
NIT	2.1E+00	1.3E+02	1.5E-02		1.3E-01	1.3E+02	9.6E-04		9.5E-02	1.3E+02	7.1E-04	
SN	2.9E+00	1.0E-01	2.9E+01		1.8E-01	3.5E+00	5.1E-02		1.4E-01	1.0E-01	1.4E+00	
ZN	2.6E+03	1.6E+02	1.6E+01		1.7E+02	1.6E+02	1.0E+00		2.1E+02	1.6E+02	1.3E+00	
DNBP	7.0E+00	6.0E+02	1.2E-02		3.9E-01	6.0E+02	6.5E-04		3.7E-01	6.0E+02	6.2E-04	
DPA	1.9E+00	3.1E+01	6.3E-02		1.2E-01	3.1E+01	3.9E-03		9.4E-02	3.1E+01	3.0E-03	
NC	5.2E+02	9.0E+03	5.7E-02		3.2E+01	9.0E+03	3.5E-03		2.4E+01	9.0E+03	2.6E-03	
CH2Cl2	1.8E-02	5.3E+01	3.3E-04		1.1E-03	5.3E+01	2.1E-05		8.5E-04	5.3E+01	1.6E-05	
B2EHP	5.1E-01	1.9E+01	2.7E-02		2.8E-02	1.9E+01	1.5E-03		2.7E-02	1.9E+01	1.4E-03	
CL	2.2E+00	6.0E+01	3.7E-02		1.4E-01	6.0E+01	2.3E-03		1.0E-01	6.0E+01	1.7E-03	
DNOP	1.0E+00	1.8E+02	5.7E-03		5.6E-02	1.8E+02	3.2E-04		5.3E-02	1.8E+02	3.0E-04	
SUMMARY HAZARD INDEX				4.6E+03	1.5E+01				2.3E+02			



TABLE R-50

ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

SPOILS DISPOSAL AREA 4, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Red fox			Red-tailed hawk		
	TBD	RTV	HI	TBD	RTV	HI
SO4	8.5E-03	1.2E+02	7.1E-05	1.2E-02	1.2E+02	9.8E-05
PB	1.5E-01	3.0E+00	5.0E-02	1.9E-01	2.5E+00	7.7E-02
24DNT	6.6E-04	1.0E+00	6.6E-04	1.1E-03	4.0E+01	2.7E-05
NIT	7.3E-04	1.3E+02	5.5E-06	1.0E-03	1.3E+02	7.6E-06
SN	1.5E-03	1.0E-01	1.5E-02	2.5E-03	3.5E+00	7.2E-04
ZN	8.2E+00	1.6E+02	5.1E-02	1.5E+01	1.6E+02	9.4E-02
DNBP	3.5E-03	6.0E+02	5.8E-06	5.8E-03	6.0E+02	9.7E-06
DPA	1.0E-03	2.5E+01	4.1E-05	1.7E-03	3.1E+01	5.4E-05
NC	1.8E-01	9.0E+03	2.0E-05	2.5E-01	9.0E+03	2.8E-05
CH2Cl2	9.4E-06	5.3E+01	1.8E-07	1.5E-05	5.3E+01	2.9E-07
B2EHP	2.5E-04	1.9E+01	1.3E-05	4.2E-04	1.9E+01	2.2E-05
CL	7.9E-04	6.0E+01	1.3E-05	1.1E-03	6.0E+01	1.8E-05
DNOP	5.0E-04	1.8E+02	2.9E-06	8.3E-04	1.8E+02	4.7E-06
SUMMARY HAZARD INDEX			1.2E-01			1.7E-01

NOTES: iHD = Total Body Dose (mg/kgBW-day)

BW = Body Weight (kg)

RTV = Reference Toxicity Value (mg/kgBW-day)

HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-51

## ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## SPOILS DISPOSAL AREA 5, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Short-tailed shrew</i>			<i>Eastern meadowlark</i>			<i>Garter snake</i>		
	TBD	RTV	HI	TBD	RTV	HI	TBD	RTV	HI
SO4	6.5E+00	1.2E+03	5.4E-03	4.0E-01	1.2E+03	3.4E-04	3.0E-01	1.2E+03	2.5E-04
PB	3.8E+02	2.0E+00	1.9E+02	2.1E+01	4.9E+00	4.3E+00	1.9E+01	2.0E+00	9.5E+00
NIT	3.1E+00	1.3E+03	2.3E-03	1.9E-01	1.3E+03	1.4E-04	1.4E-01	1.3E+03	1.1E-04
SN	3.4E+00	3.8E+01	9.1E-02	2.1E-01	3.5E+01	6.1E-03	1.7E-01	3.5E+01	4.7E-03
ZN	3.9E+03	5.0E+02	7.8E+00	2.5E+02	5.0E+02	5.0E-01	3.1E+02	5.0E+02	6.2E-01
DNBP	1.0E+01	6.0E+03	1.7E-03	5.8E-01	6.0E+03	9.6E-05	5.5E-01	6.0E+03	9.1E-05
DPA	4.2E+00	3.1E+02	1.4E-02	2.6E-01	3.1E+02	8.5E-04	2.0E-01	3.1E+02	6.6E-04
NC	1.9E+03	9.0E+04	2.1E-02	1.2E+02	9.0E+04	1.3E-03	8.7E+01	9.0E+04	9.6E-04
CH2Cl2	1.8E-02	5.3E+02	3.3E-05	1.1E-03	5.3E+02	2.1E-06	8.5E-04	5.3E+02	1.6E-06
BR	2.7E+00	7.0E+02	3.9E-03	1.7E-01	7.0E+02	2.4E-04	1.3E-01	7.0E+02	1.8E-04
CL	3.1E+00	6.0E+02	5.2E-03	1.9E-01	6.0E+02	3.2E-04	1.4E-01	6.0E+02	2.4E-04
DNOP	3.2E-01	1.8E+03	1.8E-04	1.8E-02	1.8E+03	1.0E-05	1.7E-02	1.8E+03	9.6E-06
SUMMARY HAZARD INDEX			2.0E+02			4.9E+00			1.0E+01

TABLE R-51  
ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 5, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Red fox			Red-tailed hawk		
	TBD	RTV	HI	TBD	RTV	HI
SO4	1.2E-01	1.2E+03	9.7E-05	3.2E-01	1.2E+03	2.7E-04
PB	6.3E+00	3.0E+01	2.1E-01	1.6E+01	2.5E+01	6.6E-01
NIT	5.5E-02	1.3E+03	4.1E-05	1.5E-01	1.3E+03	1.1E-04
SN	9.1E-02	3.8E+01	2.4E-03	3.0E-01	3.5E+01	8.5E-03
ZN	6.1E+02	5.0E+02	1.2E+00	2.3E+03	5.0E+02	4.5E+00
DNBP	2.6E-01	6.0E+03	4.3E-05	8.6E-01	6.0E+03	1.4E-04
DPA	1.1E-01	2.5E+02	4.5E-04	3.7E-01	3.1E+02	1.2E-03
NC	3.4E+01	9.0E+04	3.7E-04	9.3E+01	9.0E+04	1.0E-03
CH2Cl2	4.7E-04	5.3E+02	8.9E-07	1.5E-03	5.3E+02	2.9E-06
BR	4.9E-02	7.0E+02	7.0E-05	1.3E-01	7.0E+02	1.9E-04
CL	5.5E-02	6.0E+02	9.2E-05	1.5E-01	6.0E+02	2.5E-04
DNOP	7.9E-03	1.8E+03	4.5E-06	2.6E-02	1.8E+03	1.5E-05
SUMMARY HAZARD INDEX			1.4E+00			5.2E+00

NOTES: TBD = Total Body Dose (mg/kgBW-day)      BW = Body Weight (kg)  
RTV = Reference Toxicity Value (mg/kgBW-day)      HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-52  
ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 5, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Short-tailed shrew			Eastern meadowlark			Garter snake		
	TBD	RTV	HI	TBD	RTV	HI	TBD	RTV	HI
SO4	6.5E+00	1.2E+02	5.4E-02	4.0E-01	1.2E+02	3.4E-03	3.0E-01	1.2E+02	2.5E-03
PB	3.8E+02	1.0E-01	3.8E+03	2.1E+01	1.8E+00	1.2E+01	1.9E+01	1.0E-01	1.9E+02
NIT	3.1E+00	1.3E+02	2.3E-02	1.9E-01	1.3E+02	1.4E-03	1.4E-01	1.3E+02	1.1E-03
SN	3.4E+00	1.0E-01	3.4E+01	2.1E-01	3.5E+00	6.1E-02	1.7E-01	1.0E-01	1.7E+00
ZN	3.9E+03	1.6E+02	2.4E+01	2.5E+02	1.6E+02	1.6E+00	3.1E+02	1.6E+02	1.9E+00
DNBP	1.0E+01	6.0E+02	1.7E-02	5.8E-01	6.0E+02	9.6E-04	5.5E-01	6.0E+02	9.1E-04
DPA	4.2E+00	3.1E+01	1.4E-01	2.6E-01	3.1E+01	8.5E-03	2.0E-01	3.1E+01	6.6E-03
NC	1.9E+03	9.0E+03	2.1E-01	1.2E+02	9.0E+03	1.3E-02	8.7E+01	9.0E+03	9.6E-03
CH2Cl2	1.8E-02	5.3E+01	3.3E-04	1.1E-03	5.3E+01	2.1E-05	8.5E-04	5.3E+01	1.6E-05
BR	2.7E+00	7.0E+01	3.9E-02	1.7E-01	7.0E+01	2.4E-03	1.3E-01	7.0E+01	1.8E-03
CL	3.1E+00	6.0E+01	5.2E-02	1.9E-01	6.0E+01	3.2E-03	1.4E-01	6.0E+01	2.4E-03
DNOP	3.2E-01	1.8E+02	1.8E-03	1.8E-02	1.8E+02	1.0E-04	1.7E-02	1.8E+02	9.6E-05
SUMMARY HAZARD INDEX			3.9E+03			1.4E+01			1.9E+02

TABLE R-52  
ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
SPOILS DISPOSAL AREA 5, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Red fox			Red-tailed hawk		
	TBD	RTV	HI	TBD	RTV	HI
SO4	2.3E-03	1.2E+02	1.9E-05	3.2E-03	1.2E+02	2.7E-05
PB	1.3E-01	3.0E+00	4.2E-02	1.6E-01	2.5E+00	6.6E-02
NIT	1.1E-03	1.3E+02	8.3E-06	1.5E-03	1.3E+02	1.1E-05
SN	1.8E-03	1.0E-01	1.8E-02	3.0E-03	3.5E+00	8.5E-04
ZN	1.2E+01	1.6E+02	7.7E-02	2.3E+01	1.6E+02	1.4E-01
DNBP	5.2E-03	6.0E+02	8.6E-06	8.6E-03	6.0E+02	1.4E-05
DPA	2.3E-03	2.5E+01	9.0E-05	3.7E-03	3.1E+01	1.2E-04
NC	6.7E-01	9.0E+03	7.5E-05	9.3E-01	9.0E+03	1.0E-04
CH2Cl2	9.4E-06	5.3E+01	1.8E-07	1.5E-05	5.3E+01	2.9E-07
BR	9.8E-04	7.0E+01	1.4E-05	1.3E-03	7.0E+01	1.9E-05
CL	1.1E-03	6.0E+01	1.8E-05	1.5E-03	6.0E+01	2.5E-05
DNOP	1.6E-04	1.8E+02	9.1E-07	2.6E-04	1.8E+02	1.5E-06
SUMMARY HAZARD INDEX			1.4E-01			2.1E-01

NOTES: TBD = Total Body Dose (mg/kgBW-day)      BW = Body Weight (kg)  
RTV = Reference Toxicity Value (mg/kgBW-day)      HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-53

## ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## ROCKET PASTE AREA, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Short-tailed shrew			Eastern meadowlark			Garter snake		
	TBD	RTV	HI	TBD	RTV	HI	TBD	RTV	HI
24DNT	1.4E+03	5.4E+01	2.6E+01	8.8E+01	5.4E+01	1.6E+00	6.9E+01	5.4E+01	1.3E+00
26DNT	5.7E+01	5.4E+01	1.1E+00	3.5E+00	5.4E+01	6.6E-02	2.8E+00	5.4E+01	5.1E-02
BAANTR	1.1E+00	2.0E+01	5.3E-02	5.8E-02	2.0E+01	2.9E-03	5.6E-02	2.0E+01	2.8E-03
CR	3.8E+01	6.0E+01	6.3E-01	2.3E+00	2.5E+01	9.0E-02	1.9E+00	2.5E+01	7.6E-02
DEP	8.4E+01	1.7E+03	4.9E-02	4.9E+00	1.7E+03	2.9E-03	4.2E+00	1.7E+03	2.4E-03
HG	5.5E-01	3.6E+00	1.5E-01	3.9E-02	4.0E-01	9.9E-02	3.1E-02	4.0E-01	7.7E-02
NG	2.6E+03	3.2E+02	8.4E+00	1.6E+02	3.2E+02	5.2E-01	1.3E+02	3.2E+02	4.1E-01
NNDPA	1.7E+04	5.0E+02	3.4E+01	1.0E+03	5.0E+02	2.0E+00	8.5E+02	5.0E+02	1.7E+00
PB	1.3E+04	2.0E+00	6.5E+03	7.2E+02	4.9E+00	1.5E+02	6.5E+02	2.0E+00	3.2E+02
PYR	1.5E+00	1.6E+02	9.3E-03	8.3E-02	1.6E+02	5.2E-04	7.8E-02	1.6E+02	4.9E-04
123PDA	3.3E+01	8.0E+03	4.2E-03	2.1E+00	8.0E+03	2.6E-04	1.6E+00	8.0E+03	2.0E-04
CHRY	1.6E+00	9.9E+02	1.6E-03	8.8E-02	9.9E+02	8.9E-05	8.4E-02	9.9E+02	8.5E-05
FANT	1.8E+00	4.0E+02	4.5E-03	9.9E-02	4.0E+02	2.5E-04	9.4E-02	4.0E+02	2.4E-04
NIT	2.1E+01	1.3E+03	1.5E-02	1.3E+00	1.3E+03	9.6E-04	9.5E-01	1.3E+03	7.1E-04
NNDMEA	5.3E-01	4.6E+00	1.2E-01	3.3E-02	4.6E+00	7.2E-03	2.6E-02	4.6E+00	5.6E-03
NNDNPA	4.1E-01	5.1E+01	7.9E-03	2.5E-02	5.1E+01	4.9E-04	2.0E-02	5.1E+01	3.8E-04
PHANTR	4.5E-01	1.4E+02	3.2E-03	2.5E-02	1.4E+02	1.8E-04	2.3E-02	1.4E+02	1.7E-04
SO4	3.9E+00	1.2E+03	3.3E-03	2.4E-01	1.2E+03	2.0E-04	1.8E-01	1.2E+03	1.5E-04
SUMMARY HAZARD INDEX				6.6E+03			1.5E+02		
							3.3E+02		

TABLE R-53  
ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
ROCKET PASTE AREA, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Red fox			Red-tailed hawk		
	TBD	RTV	HI	TBD	RTV	HI
24DNT	3.8E+01	5.0E+00	7.6E+00	1.2E+02	5.4E+01	2.3E+00
26DNT	1.5E+00	5.0E+00	3.1E-01	5.0E+00	5.4E+01	9.2E-02
BAANTR	2.6E-02	2.0E+01	1.3E-03	8.7E-02	2.0E+01	4.4E-03
CR	1.1E+00	6.0E+01	1.9E-02	3.8E+00	2.5E+01	1.5E-01
DEP	2.2E+00	1.7E+03	1.3E-03	7.1E+00	1.7E+03	4.1E-03
HG	6.6E-02	1.0E+00	6.6E-02	2.3E-01	4.0E-01	5.7E-01
NG	7.0E+01	2.5E+01	2.8E+00	2.3E+02	3.2E+02	7.3E-01
NNDPA	4.4E-02	5.0E+02	8.8E-01	1.4E+03	5.0E+02	2.9E+00
PB	2.2E+02	3.0E+01	7.2E+00	5.6E+02	2.5E+01	2.2E+01
PYR	3.7E-02	1.6E+02	2.3E-04	1.2E-01	1.6E+02	7.7E-04
123PDA	8.9E-01	8.0E+03	1.1E-04	2.9E+00	8.0E+03	3.6E-04
CHRY	3.9E-02	9.9E+02	4.0E-05	1.3E-01	9.9E+02	1.3E-04
FANT	4.5E-02	4.0E+02	1.1E-04	1.5E-01	4.0E+02	3.7E-04
NIT	3.7E-01	1.3E+03	2.8E-04	1.0E+00	1.3E+03	7.6E-04
NNDMEA	1.4E-02	2.5E+01	5.7E-04	4.6E-02	4.6E+00	1.0E-02
NNDNPA	1.1E-02	5.1E+01	2.1E-04	3.5E-02	5.1E+01	6.9E-04
PHANTR	1.1E-02	1.4E+02	8.0E-05	3.7E-02	1.4E+02	2.7E-04
SO4	7.0E-02	1.2E+03	5.8E-05	1.9E-01	1.2E+03	1.6E-04
SUMMARY HAZARD INDEX			1.9E+01	2.9E+01		

NOTES: TBD = Total Body Dose (mg/kgBW-day)      BW = Body Weight (kg)  
RTV = Reference Toxicity Value (mg/kgBW-day)      HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-54  
ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
ROCKET PASTE AREA, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Short-tailed shrew			Eastern meadowlark			Garter snake		
	TBD	RTV	HI	TBD	RTV	HI	TBD	RTV	HI
24DNT	1.4E+03	4.0E+01	3.6E+01	8.8E+01	4.0E+01	2.2E+00	6.9E+01	4.0E+01	1.7E+00
26DNT	5.7E+01	4.0E+01	1.4E+00	3.5E+00	4.0E+01	8.9E-02	2.8E+00	4.0E+01	6.9E-02
BAANTR	1.1E+00	2.0E+00	5.3E-01	5.8E-02	2.0E+00	2.9E-02	5.6E-02	2.0E+00	2.8E-02
CR	3.8E+01	5.7E+00	6.6E+00	2.3E+00	3.5E+00	6.5E-01	1.9E+00	3.5E+00	5.4E-01
DEP	8.4E+01	1.7E+02	4.9E-01	4.9E+00	1.7E+02	2.9E-02	4.2E+00	1.7E+02	2.5E-02
HG	5.5E-01	1.2E-01	4.6E+00	3.9E-02	7.0E-03	5.6E+00	3.1E-02	7.0E-03	4.4E+00
NG	2.6E+03	3.2E+01	8.3E+01	1.6E+02	3.2E+01	5.1E+00	1.3E+02	3.2E+01	4.0E+00
NNDPA	1.7E+04	5.0E+01	3.4E+02	1.0E+03	5.0E+01	2.0E+01	8.5E+02	5.0E+01	1.7E+01
PB	1.3E+04	1.0E-01	1.3E+05	7.2E+02	1.8E+00	4.0E+02	6.5E+02	1.0E-01	6.5E+03
PYR	1.5E+00	1.3E+02	1.1E-02	8.3E-02	1.3E+02	6.4E-04	7.8E-02	1.3E+02	6.0E-04
123PDA	3.3E+01	8.0E+02	4.2E-02	2.1E+00	8.0E+02	2.6E-03	1.6E+00	8.0E+02	2.0E-03
CHRY	1.6E+00	9.9E+01	1.6E-02	8.8E-02	9.9E+01	8.9E-04	8.4E-02	9.9E+01	8.5E-04
FANT	1.8E+00	4.0E+01	4.5E-02	9.9E-02	4.0E+01	2.5E-03	9.4E-02	4.0E+01	2.4E-03
NIT	2.1E+01	1.3E+02	1.5E-01	1.3E+00	1.3E+02	9.6E-03	9.5E-01	1.3E+02	7.1E-03
NNDMEA	5.3E-01	4.6E-01	1.2E+00	3.3E-02	4.6E-01	7.2E-02	2.6E-02	4.6E-01	5.6E-02
NNDNPA	4.1E-01	5.1E+00	7.9E-02	2.5E-02	5.1E+00	4.9E-03	2.0E-02	5.1E+00	3.8E-03
PHANTR	4.5E-01	1.4E+01	3.2E-02	2.5E-02	1.4E+01	1.8E-03	2.3E-02	1.4E+01	1.7E-03
SO4	3.9E+00	1.2E+02	3.3E-02	2.4E-01	1.2E+02	2.0E-03	1.8E-01	1.2E+02	1.5E-03
SUMMARY HAZARD INDEX			1.3E+05			4.4E+02			6.5E+03



TABLE R-54  
ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
ROCKET PASTE AREA, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Red fox</i>			<i>Red-tailed hawk</i>		
	TBD	RTV	HI	TBD	RTV	HI
24DNT	3.8E+01	1.0E+00	3.8E+01	1.2E+02	4.0E+01	3.1E+00
26DNT	1.5E+00	1.0E+00	1.5E+00	5.0E+00	4.0E+01	1.2E-01
BAANTR	2.6E-02	2.0E+00	1.3E-02	8.7E-02	2.0E+00	4.4E-02
CR	1.1E+00	5.7E+00	2.0E-01	3.8E+00	3.5E+00	1.1E+00
DEP	2.2E+00	1.7E+02	1.3E-02	7.1E+00	1.7E+02	4.2E-02
HG	6.6E-02	1.0E-01	6.6E-01	2.3E-01	7.0E-03	3.2E+01
NG	7.0E+01	3.0E+00	2.3E+01	2.3E+02	3.2E+01	7.2E+00
NNDPA	4.4E+02	5.0E+01	8.8E+00	1.4E+03	5.0E+01	2.9E+01
PB	2.2E+02	3.0E+00	7.2E+01	5.6E+02	2.5E+00	2.2E+02
PYR	3.7E-02	1.3E+02	2.9E-04	1.2E-01	1.3E+02	9.5E-04
123PDA	8.9E-01	8.0E+02	1.1E-03	2.9E+00	8.0E+02	3.6E-03
CHRY	3.9E-02	9.9E+01	4.0E-04	1.3E-01	9.9E+01	1.3E-03
FANT	4.5E-02	4.0E+01	1.1E-03	1.5E-01	4.0E+01	3.7E-03
NIT	3.7E-01	1.3E+02	2.8E-03	1.0E+00	1.3E+02	7.6E-03
NNDMEA	1.4E-02	2.5E+00	5.7E-03	4.6E-02	4.6E-01	1.0E-01
NNDNPA	1.1E-02	5.1E+00	2.1E-03	3.5E-02	5.1E+00	6.9E-03
PHANTR	1.1E-02	1.4E+01	8.0E-04	3.7E-02	1.4E+01	2.7E-03
SO4	7.0E-02	1.2E+02	5.8E-04	1.9E-01	1.2E+02	1.6E-03
SUMMARY HAZARD INDEX			1.4E+02	3.0E+02		

NOTES: TBD = Total Body Dose (mg/kgBW-day)      BW = Body Weight (kg)  
RTV = Reference Toxicity Value (mg/kgBW-day)      HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-55  
ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
NITROGLYCERINE POND, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Short-tailed shrew			Eastern meadowlark			Garter snake		
	TBD	RTV	HI	TBD	RTV	HI	TBD	RTV	HI
HG	1.9E+00	3.6E+00	5.2E-01	1.3E-01	4.0E-01	3.3E-01	9.7E-02	4.0E-01	2.4E-01
NG	2.8E+01	3.2E+02	8.8E-02	1.7E+00	3.2E+02	5.5E-03	1.3E+00	3.2E+02	4.1E-03
NH3	3.0E+00	4.8E+01	6.3E-02	1.9E-01	4.8E+01	3.9E-03	1.4E-01	4.8E+01	2.9E-03
PB	3.8E+04	2.0E+00	1.9E+04	2.1E+03	4.9E+00	4.3E+02	1.9E+03	2.0E+00	9.3E+02
SUMMARY HAZARD INDEX			1.9E+04			4.3E+02			9.3E+02

TABLE R-55  
ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
NITROGLYCERINE POND, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Red fox			Red-tailed hawk		
	TBD	RTV	HI	TBD	RTV	HI
HG	1.6E-01	1.0E+00	1.6E-01	5.9E-01	4.0E-01	1.5E+00
NG	6.3E-01	2.5E+01	2.5E-02	2.0E+00	3.2E+02	6.3E-03
NH3	5.4E-02	3.2E+03	1.7E-05	1.5E-01	4.8E+01	3.1E-03
Pb	5.0E+02	3.0E+01	1.7E+01	1.2E+03	2.5E+01	4.9E+01
SUMMARY HAZARD INDEX			1.7E+01	5.0E+01		

NOTES: TBD = Total Body Dose (mg/kgBW-day)      BW = Body Weight (kg)  
RTV = Reference Toxicity Value (mg/kgBW-day)      HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-56

## ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## NITROGLYCERINE POND, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Short-tailed shrew				Eastern meadowlark				Garter snake			
	TBD	RTV	HI	HI	TBD	RTV	HI	HI	TBD	RTV	HI	HI
HG	1.9E+00	1.2E-01	1.5E+01	1.5E+01	5.3E-02	7.0E-03	7.6E+00	7.6E+00	3.9E-02	7.0E-03	5.5E+00	5.5E+00
NO	2.8E+01	3.2E+01	8.8E-01	8.8E-01	6.9E-01	3.2E+01	2.2E-02	2.2E-02	5.2E-01	3.2E+01	1.7E-02	1.7E-02
NH3	3.0E+00	2.0E+01	1.5E-01	1.5E-01	7.5E-02	2.0E+01	3.8E-03	3.8E-03	5.6E-02	2.0E+01	2.8E-03	2.8E-03
PB	3.8E+04	1.0E-01	3.8E+05	3.8E+05	8.4E+02	1.8E+00	4.8E+02	4.8E+02	7.4E+02	1.0E-01	7.4E+03	7.4E+03
SUMMARY HAZARD INDEX				3.8E+05					4.8E+02			

TABLE R-56  
ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
NITROGLYCERINE POND, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Red fox</i>			<i>Red-tailed hawk</i>		
	TBD	RTV	HI	TBD	RTV	HI
HG	1.3E-03	1.0E-01	1.3E-02	2.4E-03	7.0E-03	3.4E-01
NG	5.0E-03	3.0E+00	1.7E-03	7.9E-03	3.2E+01	2.5E-04
NH3	4.3E-04	3.2E+02	1.4E-06	6.0E-04	2.0E+01	3.0E-05
PB	4.0E+00	3.0E+00	1.3E+00	4.9E+00	2.5E+00	2.0E+00
SUMMARY HAZARD INDEX				1.3E+00		2.3E+00

NOTES: TBD = Total Body Dose (mg/kgBW-day)      BW = Body Weight (kg)  
RTV = Reference Toxicity Value (mg/kgBW-day)      HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-57

## ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

 REMEDIAL INVESTIGATION  
 OLEUM PLANT POND, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Short-tailed shrew</i>			<i>Eastern meadowlark</i>			<i>Garter snake</i>		
	TBD	RTV	HI	TBD	RTV	HI	TBD	RTV	HI
SO <sub>4</sub>	1.5E+03	1.2E+03	1.2E+00	9.0E+01	1.2E+03	7.6E-02	6.7E+01	1.2E+03	5.6E-02
NIT	5.9E-01	1.3E+03	4.5E-04	3.7E-02	1.3E+03	2.8E-05	2.7E-02	1.3E+03	2.0E-05
SUMMARY HAZARD INDEX									
			1.2E+00			7.6E-02			5.6E-02

TABLE R-57  
ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
OLEUM PLANT POND, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Red fox</i>			<i>Red-tailed hawk</i>		
	TBD	RTV	HI	TBD	RTV	HI
SO4	2.6E+01	1.2E+03	2.2E-02	7.2E+01	1.2E+03	6.0E-02
NIT	1.1E-02	1.3E+03	7.9E-06	2.9E-02	1.3E+03	2.2E-05
SUMMARY HAZARD INDEX			2.2E-02	6.0E-02		

NOTES: TBD = Total Body Dose (mg/kgBW-day)  
RTV = Reference Toxicity Value (mg/kgBW-day)  
BW = Body Weight (kg)  
HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-58

ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

OLEUM PLANT POND, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Short-tailed shrew</i>			<i>Eastern meadowlark</i>			<i>Gopher snake</i>		
	TBD	RTV	HI	TBD	RTV	HI	TBD	RTV	HI
SO <sub>4</sub>	1.5E+03	1.2E+02	1.2E+01	8.4E+01	1.2E+02	7.0E-01	6.2E+01	1.2E+02	5.2E-01
NIT	5.9E-01	1.3E+02	4.5E-03	3.4E-02	1.3E+02	2.6E-04	2.5E-02	1.3E+02	1.9E-04
SUMMARY HAZARD INDEX									
			1.2E+01			7.0E-01			5.2E-01



TABLE R-58  
ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
OLEUM PLANT POND, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Red fox		Red-tailed hawk	
	TBD	RTV	HI	HI
SO4	4.8E-01	1.2E+02	4.0E-03	5.6E-03
NIT	2.0E-04	1.3E+02	1.5E-06	2.0E-06
SUMMARY HAZARD INDEX				
			4.0E-03	5.6E-03

NOTES: TBD = Total Body Dose (mg/kgBW-day)      BW = Body Weight (kg)  
RTV = Reference Toxicity Value (mg/kgBW-day)      HI = Hazard Index (calculated by dividing TBD by RTV)

TABLE R-59  
ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
OLD ACID AREA, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Short-tailed shrew			Eastern meadowlark			Garter snake		
	TBD	RTV	HI	TBD	RTV	HI	TBD	RTV	HI
NI	1.9E+02	1.3E+01	1.5E+01	1.3E+01	1.0E+02	1.3E-01	8.4E+00	1.3E+01	6.3E-01
NIT	3.1E-01	1.3E+03	2.3E-04	1.9E-02	1.3E+03	1.4E-05	1.4E-02	1.3E+03	1.1E-05
PB	5.6E+03	2.0E+00	2.8E+03	3.1E+02	4.9E+00	6.3E+01	2.8E+02	2.0E+00	1.4E+02
SO4	3.1E+03	1.2E+03	2.6E+00	1.9E+02	1.2E+03	1.6E-01	1.4E+02	1.2E+03	1.2E-01
SUMMARY HAZARD INDEX									
			2.8E+03				6.3E+01		
			</						

TABLE R-59

## ESTIMATION OF ACUTE RISKS TO TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

## REMEDIAL INVESTIGATION

## OLD ACID AREA, BADGER ARMY AMMUNITION PLANT

CHEMICAL	Short-tailed shrew			Eastern meadowlark			Garter snake		
	TBD	RTV	HI	TBD	RTV	HI	TBD	RTV	HI
NI NIT PB SO4	1.9E+02	1.3E+01	1.5E+01	1.3E+01	1.0E+02	1.3E-01	8.4E+00	1.3E+01	6.3E-01
	3.1E-01	1.3E+03	2.3E-04	1.9E-02	1.3E+03	1.4E-05	1.4E-02	1.3E+03	1.1E-05
	5.6E+03	2.0E+00	2.8E+03	3.1E+02	4.9E+00	6.3E+01	2.8E+02	2.0E+00	1.4E+02
	3.1E+03	1.2E+03	2.6E+00	1.9E+02	1.2E+03	1.6E-01	1.4E+02	1.2E+03	1.2E-01
SUMMARY HAZARD INDEX									
			2.8E+03			6.3E+01			1.4E+02

TABLE R-60  
ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION

REMEDIAL INVESTIGATION  
OLD ACID AREA, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Short-tailed shrew</i>			<i>Eastern meadowlark</i>			<i>Garter snake</i>		
	TBD	RTV	HI	TBD	RTV	HI	TBD	RTV	HI
SO4	3.1E+03	1.2E+02	2.6E+01	1.9E+02	1.2E+02	1.6E+00	1.4E+02	1.2E+02	1.2E+00
PB	5.6E+03	1.0E-01	5.6E+04	3.1E+02	1.8E+00	1.8E+02	2.8E+02	1.0E-01	2.8E+03
NI	1.9E+02	1.3E+00	1.5E+02	1.3E+01	1.0E+01	1.3E+00	8.4E+00	1.3E+00	6.5E+00
NIT	3.1E-01	1.3E+02	2.3E-03	1.9E-02	1.3E+02	1.5E-04	1.4E-02	1.3E+02	1.1E-04
SUMMARY HAZARD INDEX									
			5.7E+04			1.8E+02			2.8E+03

TABLE R-60  
ESTIMATION OF CHRONIC RISKS TO INDIVIDUAL TERRESTRIAL ORGANISMS FROM FOOD AND SOIL INGESTION  
REMEDIAL INVESTIGATION  
OLD ACID AREA, BADGER ARMY AMMUNITION PLANT

CHEMICAL	<i>Red fox</i>			<i>Red-tailed hawk</i>		
	TBD	RTV	HI	TBD	RTV	HI
SO4	2.3E+00	1.2E+02	1.9E-02	3.1E+00	1.2E+02	2.6E-02
PB	3.9E+00	3.0E+00	1.3E+00	5.0E+00	2.5E+00	2.0E+00
NI	1.4E-01	6.3E+01	2.3E-03	1.8E-01	1.0E+01	1.8E-02
NIT	2.3E-04	1.3E+02	1.7E-06	3.1E-04	1.3E+02	2.4E-06
SUMMARY HAZARD INDEX			1.3E+00	2.0E+00		

NOTES: TBD = Total Body Dose (mg/kgBW-day)  
RTV = Reference Toxicity Value (mg/kgBW-day)  
BW = Body Weight (kg)  
HI = Hazard Index (calculated by dividing TBD by RTV)